Paid Circulation, 32,937

Annual Meeting
Hotel Statler, Boston
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The New England Journal of Medicine

FORMERLY THE BOSTON MEDICAL AND SURGICAL JOURNAL — *Established 1828*

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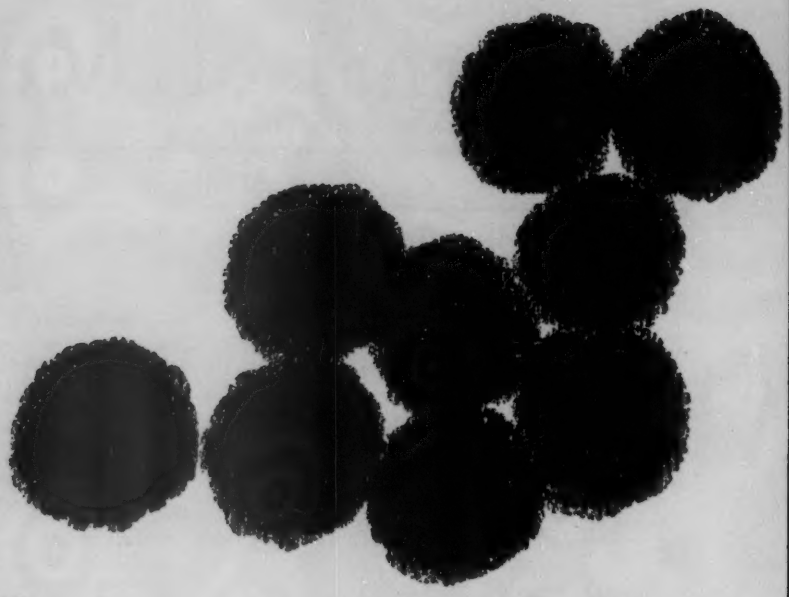

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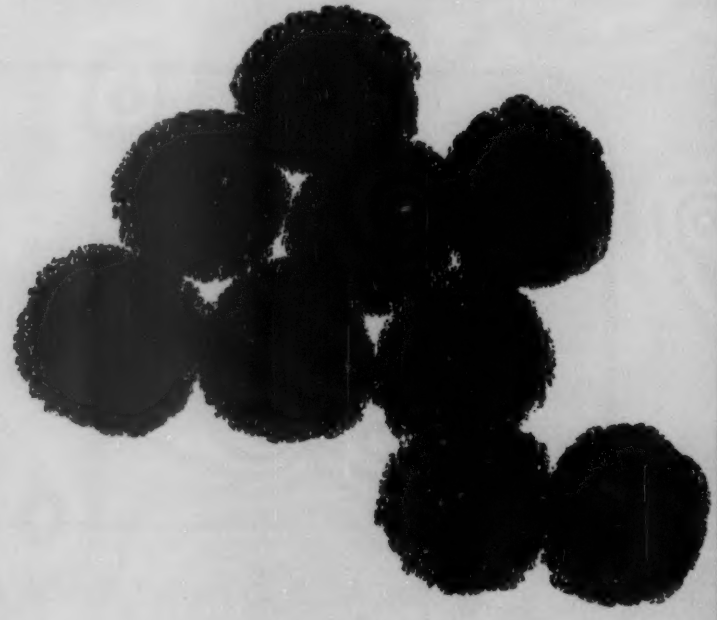

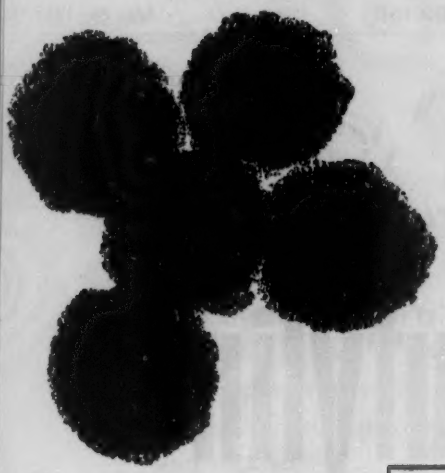

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*Pratt, R., & Dufrenoy, J.: Texas Rep. Biol. & Med. *12*:145, 1954.

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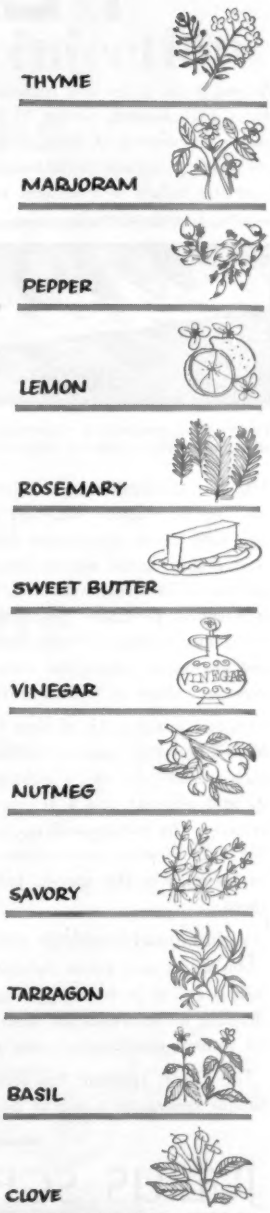

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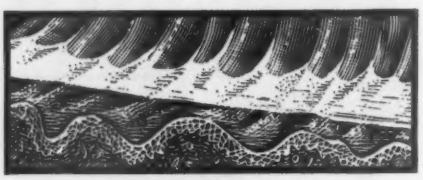

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Office treatment. Expose vagina with speculum. Wipe walls dry with cotton sponges and wash thoroughly for about three minutes with a 1:250 dilution of VAGISEC liquid. Remove excess fluid with cotton sponges. Office treatments are an integral part of the Davis technic.

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1. Davis, C.H.: Am. J. Obst. & Gynec. *68*:559 (Aug.) 1954.
2. Davis, C.H.: West. Jour. Surg. *63*:53 (Feb.) 1955.
3. Davis, C.H.: J.A.M.A. *157*:126 (Jan. 8) 1955.

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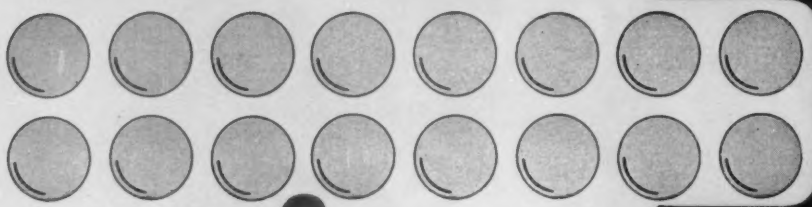

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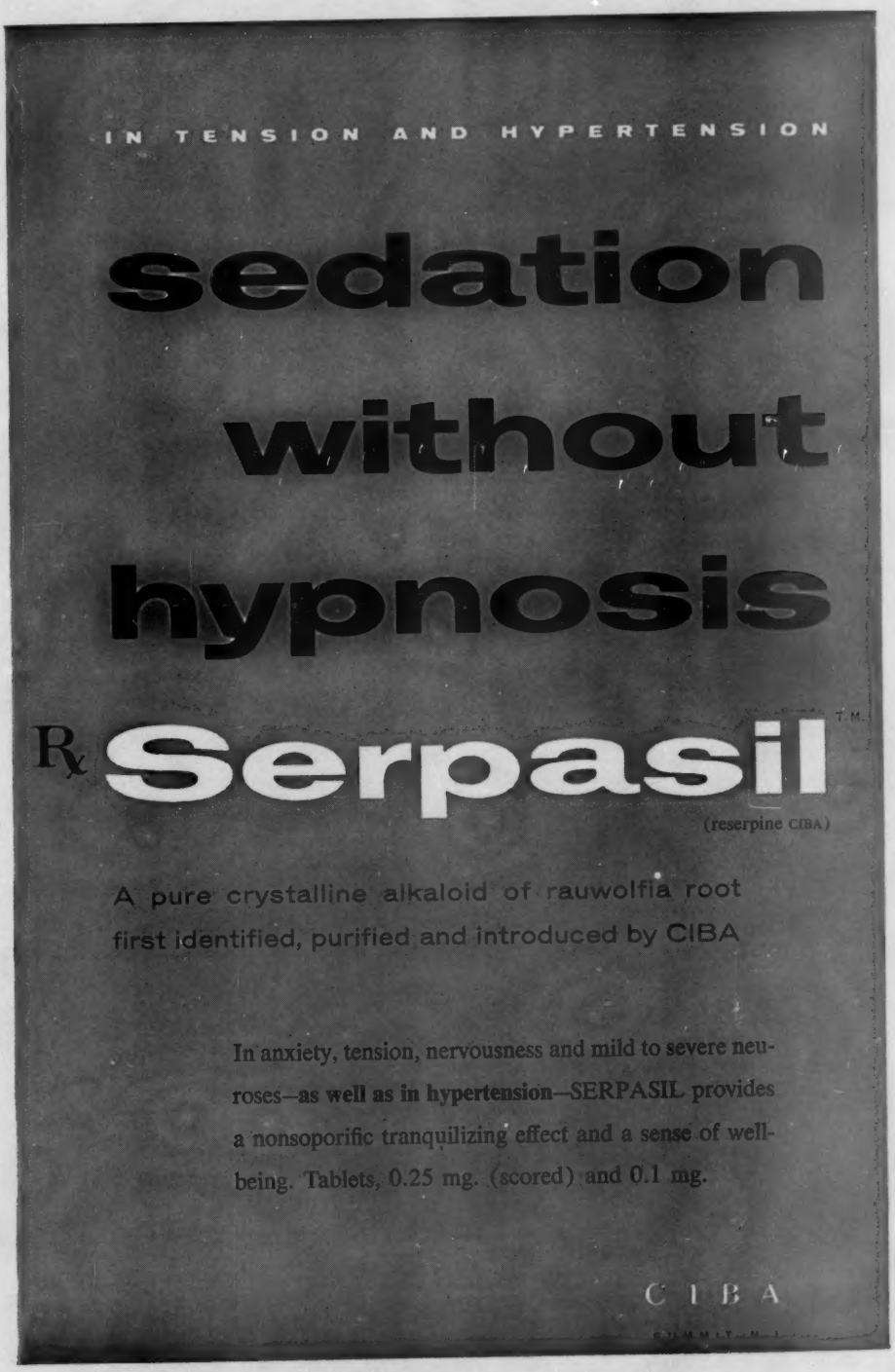
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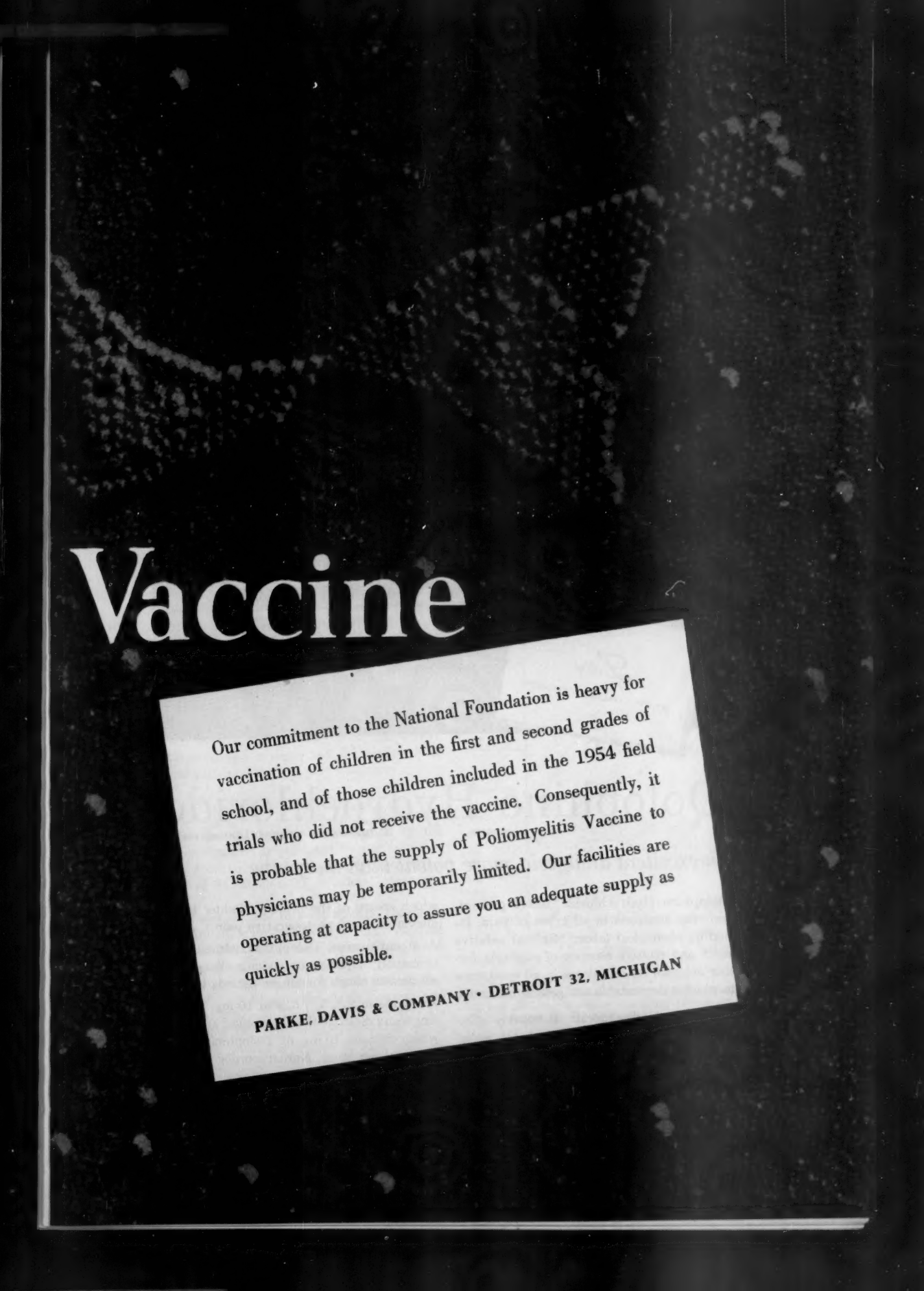
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Volume 252 APRIL 28, 1955 Number 17

COMPARATIVE STUDY OF SMOKING HABITS OF PHYSICIANS

LEONID S. SNEGIREFF, M.D.,* AND OLIVE M. LOMBARD, S.M.†

BOSTON

THE need for current data on smoking to serve as a base line for future comparisons is obvious, and the saying, "physician know thyself," applies to this effort.

Most of the tobacco today is derived from *Nicotiana tabacum*, a plant that is native to South America and Mexico. Before Columbus, *N. rustica* was commonly smoked by the Indians of Eastern North America whereas *N. attenuata, N. quadrivolvis* and *N. trigonophylla* were the popular plants with the Indian tribes of the West.

Since John Rolfe began tobacco culture in Jamestown, Virginia, in 1612, the actual processing of tobacco for smoking purposes has undergone many changes, from fire-curing in Colonial days, to the use of charcoal for curing purposes about 1825 and of a furnace with metal flues in Civil War days and on to the modern methods of curing with special emphasis on the taste of the end product and its classification into fire-cured, dark air-cured, flue-cured, Burley, Maryland and perique and the binder, filler and wrapper suitable for cigar manufacture.

TOBACCO CONSUMPTION

The importance of tobacco consumption to the economy of the nation is indicated by the fact that in 1951 the tobacco industry in the United States paid a total of $1,810,396,000 in federal, state and local taxes.

Figure 1 shows the rise in total tobacco consumption in the United States, 1896-1950. The increase is principally attributable to the phenomenal growth of cigarette smoking.

It is generally accepted that nicotine and related alkaloids of tobacco furnish the habit-forming and narcotic effects in human beings. These effects account in part for the general indulgence in smoking for the better part of a lifetime of most smokers.

*Associate professor of cancer control, Harvard School of Public Health; director, Cancer Control Unit at Cancer Research Institute, New England Deaconess Hospital.

†Instructor in public-health practice, Harvard School of Public Health; biometrician, Cancer Control Unit at Cancer Research Institute, New England Deaconess Hospital.

BIOLOGIC ASPECTS OF SMOKING

In the last few years, particularly since the wide publicity given to the rising mortality from cancer of the lung in the nation, the biologic effect of smoking has come under particular scrutiny of the medical profession.

Our special interest in arsenic and cancer[1] led us to a study of multiple factors that are capable of affecting mortality rates in the metallurgical indus-

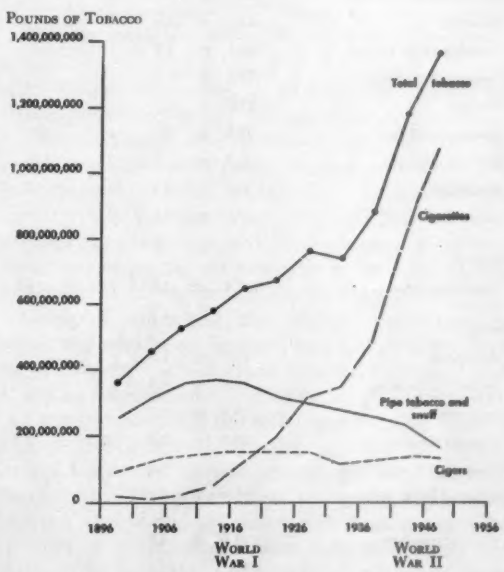

FIGURE 1. *Five-Year Averages of Pounds of Tobacco Leaf Consumed in the Manufacture of Cigars, Cigarettes, Chewing and Smoking Tobacco and Snuff (Based on the Annual Reports of the Commissioner of Internal Revenue).*

try. In 1951, together with Herbert L. Lombard, M.D., we began an epidemiologic study of lung cancer that is currently approaching its completion. In the course of collecting personal histories of apparently healthy people for "group comparison" we be-

came aware of the paucity of scientific data on smoking habits of the population by age and occupational status.

A survey was undertaken early in 1954, and a preliminary report of the smoking habits of physicians in Massachusetts has been published.[2] Since then, the report to the American Medical Association by

TABLE 1. *Percentage of Massachusetts Physicians Not Smoking, by Type of Practice.*

TYPE OF PRACTICE	PHYSICIANS NOT SMOKING	STANDARD ERROR*	AVERAGE NOT SMOKING	STANDARD ERROR*
	%	%	%	%
All types	33.5	± 0.7		
Group 1:				
Preventive medicine & public health	53.7	± 7.8		
Thoracic surgery	52.6	± 11.5		
Dermatology	44.4	± 6.3		
Neurosurgery	44.4	± 11.7		
Orthopedics (orthopedic surgery)	41.4	± 5.0		
Pediatrics	40.9	± 3.5		
Ophthalmology (alone)	40.0	± 4.9		
Pathology	39.5	± 5.6	39.1	± 1.2
Otolaryngology (alone)	38.5	± 4.8		
Research & teaching	37.5	± 12.1		
Urology	37.5	± 5.7		
Internal medicine	37.5	± 2.0		
Physical medicine	35.7	± 12.8		
Radiology	35.1	± 4.5		
Chest diseases	34.6	± 6.6		
Group 2:				
General practice	31.4	± 1.4	31.4	± 1.4
Group 3:				
Anesthesia	30.2	± 4.0		
Industrial medicine	29.5	± 6.9		
Administration	29.4	± 11.0		
General surgery	29.4	± 2.0	28.2	± 1.3
Neurology & psychiatry	28.1	± 2.8		
Gynecology & obstetrics	25.3	± 2.7		
Both ophthalmology & otolaryngology	21.6	± 6.8		
Proctology	17.6	± 9.2		
Miscellaneous	38.4	± 4.0		

*Chance variation of rate.

Cuyler Hammond and Daniel Horn, of the American Cancer Society, on the relation between smoking and cancer of the lung has further sharpened the interest of the profession in the prevalence of smoking of tobacco in its different forms.

The data reported below may serve to give a clearer insight into the relative degrees of indulgence in smoking and the duration of abstinence.

SMOKING HABITS IN RELATION TO TYPE OF MEDICAL PRACTICE

The survey of Massachusetts physicians referred to above was conducted by the Cancer Control Unit of the New England Deaconess Hospital Cancer Research Institute and approved by the Cancer Committee of the Massachusetts Medical Society. Questionnaires were distributed to the physicians requesting information about their personal smoking habits. The previous paper[2] discussed the over-all situation of smoking habits and beliefs.* The scope of the present paper extends to smoking habits in relation to the type of practice of the responding physicians.

TABLE 2. *Percentages of Physicians Who Never Smoked and of Physicians Who Stopped Smoking, by Type of Practice.*

GROUP	PHYSICIANS WHO NEVER SMOKED	STANDARD ERROR	PHYSICIANS WHO HAD STOPPED SMOKING	STANDARD ERROR
	%	%	%	%
1	17.0	±0.9	22.1	±1.0
2	13.3	±1.0	18.1	±1.1
3	12.6	±0.9	15.6	±1.0

The questionnaire included a statement of type of practice, whether general, partial or complete specialization and the particular specialty in which certification was achieved. An insufficient number of replies designated whether specialization was partial or complete to enable the records to be subdivided according to this variable. Thus, if a specialty was indicated, even though the physician might be partially specializing in the field, he was classified as being in that type of specialized practice.† No difference was found between those certified and those not certified in a specialty, so that these groups were combined. Furthermore, the areas of specialization were not always distinct. It was necessary to combine physicians specializing in gynecology with those specializing in obstetrics, for 73 per cent indicated specialization in both types of practice. Similarly, neurologists, with the exception of those listed specifically as neurosurgeons, were combined with psychiatrists, since many indicated specialization in neuropsychiatry; orthopedic

*4104 reports were received before April 1, 1954. The present paper is based on 4171 reports, the additional 67 replies being received between April 1 and May 31, 1954.

†Among those specializing in general surgery 74 indicated that they were specializing wholly, 27 indicated partial specialization and 413 gave no indication of the completeness of their specialization. It is assumed that the large majority of these unspecified reports were indicative of complete specialization, since it is believed that physicians specializing completely in a type of practice would assume that complete specialization was self-evident unless the contrary was specified.

surgeons were combined with physicians who referred to their specialty as orthopedics, and the few physicians who indicated only that they were specializing in research or in teaching were combined.

TABLE 3. *Percentage of Cigarette Smokers Among Physicians Who Smoke by Type of Practice.*

TYPE OF PRACTICE	PHYSICIANS SMOKING CIGARETTES	STANDARD ERROR*	AVERAGE SMOKING CIGARETTES	STANDARD ERROR*
	%	%	%	%
All types	78.5	± 0.8		
Group 1:				
Preventive medicine & public health	77.8	± 9.8		
Thoracic surgery	66.7	± 15.7		
Dermatology	64.7	± 8.2		
Neurosurgery	80.0	± 12.6		
Orthopedics (orthopedic surgery)	84.6	± 5.0		
Pediatrics	82.9	± 3.6		
Ophthalmology (alone)	79.3	± 5.3		
Pathology	80.0	± 6.0	79.9	± 1.3
Otolaryngology (alone)	66.1	± 6.0		
Research & teaching	80.0	± 12.6		
Urology	77.8	± 6.2		
Internal medicine	82.0	± 2.0		
Physical medicine	100.0(n=8)	—		
Radiology	78.9	± 4.8		
Chest diseases	85.3	± 6.1		
Group 2:				
General practice	75.4	± 1.5	75.4	± 1.5
Group 3:				
Anesthesia	80.5	± 4.3		
Industrial medicine	89.7	± 5.7		
Administration	83.3	± 10.8		
General surgery	81.3	± 2.1	81.7	± 1.3
Neurology & psychiatry	80.7	± 2.9		
Gynecology & obstetrics	82.4	± 2.8		
Both ophthalmology & otolaryngology	82.8	± 7.0		
Proctology	92.9	± 6.9		
Miscellaneous	58.1	± 5.3		

*Chance variation of rate.

Allergists and cardiologists were included in the group of those specializing in internal medicine.

Table 1 presents the percentage of physicians not smoking, with subdivisions by specialties. The types of practice have been combined into three main groups on the basis of the magnitude of the rates.*

*Proctology, owing to the small number of physicians with this specialty, and "miscellaneous" specialties have not been included in this grouping.

Group 1 comprised specialties with rates for not smoking that were above the average for all types (33.5 per cent). Group 2 included the general practitioners, whose rate was close to the average, and Group 3 other specialties, with rates below the average. A comparison between each of these groups and the average for all types showed that the percentage of physicians not smoking was higher in Group 1 than in the entire series, and lower in Group 3; no significant difference existed between Group 2 and the average for all types.

The percentage of physicians not smoking in Group 1 was significantly above that for either Group 2 or Group 3. The difference between Group 2 and Group 3 was not significant. It is apparent that distinct differences in smoking habits exist between physicians in the various types of practice.

TABLE 4. *Smoking Habits of Physicians, by Type of Practice.*

SMOKING HABIT	GROUP 1	GROUP 2	GROUP 3
	%	%	%
None	39.1	31.4	28.2
Cigarettes	33.6	34.7	40.7
Other	11.9	16.4	13.0
Mixed*	13.8	15.7	17.0
Unknown	1.6	1.8	1.1

*Represents combinations of cigarettes with cigars or pipe or both.

The physicians who were not smoking were divided into those who never smoked and those who had stopped smoking (Table 2). In each of the three groups the percentage of physicians who had stopped smoking appeared to be somewhat greater than that of physicians who had never smoked, but in Group 3 this difference was not significant.

Group 1 contained the highest percentage of physicians who never smoked, and Group 3 the lowest. However, the difference between Groups 2 and 3 was not significant. Similarly, for physicians who had stopped smoking, the difference between Groups 2 and 3 was not statistically significant. Again, Group 1 has the highest percentage, and Group 3 the lowest. It appears that there are differences between the physicians when classified into groups of types of practice, and that these differences are observed in both the percentages of physicians who have stopped smoking and the percentages who apparently have never smoked.

The percentages of cigarette smoking among physicians who smoked in the various types of practice are presented in Table 3. Group 2 appears to have a somewhat smaller percentage of cigarette smokers, although only the difference between Groups 2 and 3 is significant.

The distribution of smoking habits for the three groups is recorded in Table 4. When the percent-

age of cigarette smokers is based upon the total number of physicians in the respective group the difference between the three groups appears more evident, Group 1 having the smallest percentage of physicians smoking cigarettes and Group 3 the largest. Since the groups were constructed according to the percentage of physicians who were not smoking, an increase in the proportion of cigarette smokers might have been expected as the groups change from the highest percentage of nonsmokers to the lowest. Moreover, 59 per cent of the physicians reported that they accepted the statement that "heavy smoking of cigarettes may lead to lung cancer." A difference among the groups in the acceptance of this statement may present a possible partial explanation for the trend in the percentage of physicians who are cigarette smokers.

TABLE 5. *Comparative Acceptance of Possible Connection between Heavy Smoking and Lung Cancer.*

TYPE OF PRACTICE	PHYSICIANS SMOKING WHO ACCEPT CONNECTION	PHYSICIANS NOT SMOKING WHO ACCEPT CONNECTION	SMOKING	NOT SMOKING
	%	%	%*	%*
All types	56.1	65.0		
Group 1:				
Preventive medicine & public health	72.2	82.6		
Thoracic surgery	77.8	100.0		
Dermatology	55.9	50.0		
Neurosurgery	40.0	75.0		
Orthopedics (orthopedic surgery)	46.2	61.0		
Pediatrics	59.5	49.4		
Ophthalmology (alone)	39.7	45.0		
Pathology	68.9	80.0	62.0±1.6	67.5±1.9
Otolaryngology (alone)	58.1	70.0		
Research & teaching	90.0	66.7		
Urology	64.4	70.4		
Internal medicine	66.6	70.5		
Physical medicine	62.5	60.0		
Radiology	66.2	84.6		
Chest diseases	64.7	94.4		
Group 2:				
General practice	50.1	64.1	50.1±1.8	64.1±2.5
Group 3:				
Anesthesia	54.0	56.4		
Industrial medicine	48.3	92.9		
Administration	25.0	60.0		
General surgery	66.8	70.9	57.1±1.7	65.7±2.5
Neurology & psychiatry	52.1	58.7		
Gynecology & obstetrics	49.7	59.4		
Both ophthalmology & otolaryngology	48.3	87.5		
Proctology	42.9	100.0		
Miscellaneous	39.5	37.5		

*Chance variation of rate.

Attitude of Physicians

The physicians in each type of practice were divided into those who smoked and those who did not smoke. Within these the percentage who accepted the statement that "heavy smoking of cigarettes may lead to lung cancer" was obtained (Table 5). No significant difference was observed among the nonsmokers in the three groups who accepted the statement. However, among physicians who were smoking at the time of the survey, a significantly higher proportion in Group 1 accepted the possible relation between cigarette smoking and lung cancer. The percentage accepting this possibility was lowest for Group 2, but the difference between Groups 2 and 3 was not sufficient to be considered meaningful, at a 1 per cent probability criterion.

TABLE 6. *Cigarettes Smoked Daily by Cigarette Smokers.*

AMOUNT SMOKED PER DAY	GROUP 1		GROUP 2		GROUP 3	
	NO. OF PHYSICIANS	PERCENTAGE	NO. OF PHYSICIANS	PERCENTAGE	NO. OF PHYSICIANS	PERCENTAGE
Occasional cigarette	33	4.4	9	1.5	19	2.6
Less than 1 package	311	41.7	207	35.1	252	34.7
One package	212	28.5	176	29.8	232	31.9
Over 1 but no more than 2 packages	173	23.2	169	28.6	192	26.4
Over 2 packages	16	2.1	29	4.9	32	4.4
Totals	745		590		727	

COMPARISON BETWEEN GROUPS	CHI-SQUARE	SMOKERS OF MORE THAN 1 PACKAGE DAILY		
		N	DIFFERENCE	STANDARD ERROR
			%	%
1 & 2	15.0	2*	−8.2	±2.5*
1 & 3	12.3	2*	−5.4	±2.3†
2 & 3	1.3	2†	2.8	±2.6†

*Significant.
†Not significant.

Amount of Smoking

The distribution of the amount smoked by cigarette smokers in the three groups is presented in Table 6. The occasional smokers were combined with those who smoked less than one package daily, and all who smoked more than one package were grouped, to test the data by chi-square. It was

found that the distribution of the physicians, by number of cigarettes smoked, in Group 1 was significantly different from that in either of the other groups. Groups 2 and 3 were not different. It appears that Group 1 contained a smaller proportion

TABLE 7. *Cigars Smoked Weekly by Cigar Smokers.*

Cigars Smoked per Week	Group 1		Group 2		Group 3	
	No. of Physicians	Percentage	No. of Physicians	Percentage	No. of Physicians	Percentage
Occasional	42	18.5	33	12.1	51	21.9
Less than 15	113	49.8	116	42.6	107	45.9
15-28	40	17.6	54	19.9	37	15.9
29 & over	32	14.1	69	25.4	38	16.3
Totals	227		272		233	

Comparison between Groups	Chi-Square	Smokers of 29 or More Cigars per Week		
		N	Difference	Standard Error
			%	%
1 & 2	9.4	1*	−11.3	±3.5*
1 & 3	0.01	1†	− 2.2	±3.4†
2 & 3	8.9	1*	9.1	±3.6*

*Significant.
†Not significant.

of heavier cigarette smokers and consequently a larger proportion of lighter cigarette smokers than the other groups. This is confirmed by a comparison of the percentage of physicians smoking over one package of cigarettes a day.

TABLE 8. *Distribution of Pipe Smokers.*

No. of Pipefuls per Day	Group 1		Group 2		Group 3	
	No. of Physicians	Percentage	No. of Physicians	Percentage	No. of Physicians	Percentage
Occasional	47	16.7	27	12.9	51	20.4
Less than 5	131	46.5	109	51.9	114	45.6
5-15	92	32.6	63	30.0	74	29.6
16-30	10	3.5	8	3.8	8	3.2
Over 30	2	0.7	3	1.4	3	1.2
Totals	282		210		250	

Table 7 presents the distribution of physicians by group who are known to be smoking cigars. Among the cigar smokers the physicians in Group 2 smoked proportionately more cigars than those in the other groups. This is evidenced by both the chi-square test and a comparison of the pairs of rates. Groups 1 and 3 were not different in the amount of cigar smoking.

The distribution of physicians who smoked pipes is given in Table 8. No difference was observed between the three groups in respect to the amount of pipe tobacco consumed in a day.

TABLE 9. *Distribution of Advice Given by Physicians.*

Advice Given	Group 1		Group 2		Group 3	
	No. of Physicians	Percentage	No. of Physicians	Percentage	No. of Physicians	Percentage
Reduce smoking	825	54.6	738	64.5	766	62.7
Other advice	513	33.9	349	30.5	341	27.9
No advice	174	11.5	58	5.1	114	9.3
Stop smoking	537	35.5	393	34.3	345	28.3
Other advice	801	53.0	694	60.6	762	62.4
No advice	174	11.5	58	5.1	114	9.3
Use filters	205	13.6	164	14.3	180	14.7
Other advice	1133	74.9	923	80.6	927	75.9
No advice	174	11.5	58	5.1	114	9.3
Change to pipe or cigars	141	9.3	134	11.7	132	10.8
Other advice	1197	79.2	953	83.2	975	79.9
No advice	174	11.5	58	5.1	114	9.3
Unknown	64		30		36	
Total no. of physicians	1576		1175		1257	

TABLE 10. *Analysis of Advice Given.*

Advice	Pair of Groups	Difference	Standard Error
Reduce smoking	1 & 2	−9.9	±1.9*
	1 & 3	−8.2	±1.9*
	2 & 3	1.7	±2.0†
Stop smoking	1 & 2	1.2	±1.9†
	1 & 3	7.3	±1.8*
	2 & 3	6.1	±1.9*
Use filters	1 & 2	−0.8	±1.4†
	1 & 3	−1.2	±1.3†
	2 & 3	−0.4	±1.4†
Change to pipe or cigars	1 & 2	−2.4	±1.2†
	1 & 3	−1.5	±1.2†
	2 & 3	0.9	±1.3†

*Significant.
†Not significant.

Advice to Patients

The physicians were asked if they believed that heavy cigarette smokers should be advised to reduce

the amount of smoking, to give up smoking, to use filtered cigarettes or a filter or to change to pipe or cigars. Table 9 presents the percentage of physicians, by group, replying to each of these queries. Since many physicians recommended more than one possibility (that is, reducing or stopping), the data have been combined to show the *total* number of physicians giving advice. The respective pairs of groups were compared by the difference between the percentage of physicians suggesting each advice. Table 10 summarizes the results of these comparisons.

Reduction of smoking. The physicians in Group 1 were less apt to advise the reduction of smoking than those in the other groups. However, physicians in each group were advising reduction in preference to any of the other three types of advice.

Cessation of smoking. The physicians in Group 3 were less apt to advise heavy smokers to stop smoking than those in the other groups. The percentage advising heavy smokers to stop smoking was greater, in each group, than the percentage advising either the use of filters or a change to cigar or pipe smoking.

Use of filters. No difference in the advice to use filters was observed between the various groups. Filters were advised in preference to changing to pipe or cigar smoking in each group, but in Group 2 this difference was not significant.

Change to pipe or cigars. Ten per cent of the physicians gave the advice that a heavy cigarette smoker should change to cigar or pipe smoking. However, this advice was not as common as the other recommendations. No difference in the percentage advising this change was observed in the groups.

Conclusions

A survey of 4171 physicians, all of whom are members of a state medical society, reveals that a large proportion have stopped smoking; that among those engaged in various types of practice differences exist in the percentage not smoking and the percentage distribution of amount of cigarettes and amount of cigars smoked; that among physicians who were smoking at the time of the survey the proportion who accepted a possible relation between cigarette smoking and lung cancer varied with the type of practice; and that there was a peculiar cleavage of physicians in the three *type-of-practice* groups giving different advice to heavy cigarette smokers—55 per cent of Group 1, 64 per cent of Group 2 and 63 per cent of Group 3 advised a reduction of cigarette smoking, whereas 36 per cent of Group 1, 34 per cent of Group 2 and 28 per cent of Group 3 recommended that heavy smokers stop smoking.

Physicians in different types of practice are tending toward less cigarette smoking. A possible explanation for this tendency is the growing interest in the conceivable relation between cigarette smoking and lung cancer.

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MULTIPLE SCLEROSIS AND AMYOTROPHIC LATERAL SCLEROSIS (Concluded)*

Etiologic Significance of Recent Epidemiologic and Genetic Studies

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AMYOTROPHIC LATERAL SCLEROSIS

Clinical and Pathological Characteristics

Amyotrophic lateral sclerosis is used here synonymously with the term "progressive motor-system disease." With its clinical components, progressive muscular atrophy and progressive bulbar palsy, it is a disease in which slowly progressive degeneration of anterior-horn cells in the spinal cord and of motor nuclei in the brain stem is the most prominent feature. Betz cells in the motor cortex may also degenerate, with secondary degeneration of the corticospinal and corticobulbar tracts.[27] Muscular atrophy, which is secondary to lesions of the motor cells of the spinal cord, is nearly always pronounced at the time of death. The distribution of the muscle atrophy varies, but generally the distal muscles of the upper limbs are most severely involved. Microscopically, the distinctive picture of discrete groups of normal and atrophic muscle fibers in the same section is usually best noted in the moderately advanced stage of the muscle atrophy. The detailed microscopical picture of the central-nervous-system and muscle degeneration has been described by Lawyer and Netsky[28] and by Adams, Denny-Brown and Pearson.[27]

The demyelination is probably secondary to motor-neuron degeneration[28] rather than a "primary" demyelination as noted in multiple sclerosis. At times the demyelination of amyotrophic lateral sclerosis does appear to extend outward from the region of pyramidal tracts in the lateral-column region to the spinocerebellar tracts and on rare occasions also to the posterior columns, but it is yet to be determined whether such findings are indicative of some diffusing toxic agent or indicate that a metabolic process, which may be responsible for the disorder, has a wider range of action than the corticospinal tracts alone.

*The epidemiologic studies of multiple sclerosis were supported, in part, by a grant from the National Multiple Sclerosis Society; the investigations of amyotrophic lateral sclerosis in the Mariana Islands were made possible by a research grant from the Bureau of Medicine and Surgery, United States Navy.

Read in abstract at the International Congress of Clinical Pathology, Section on the Demyelinating Diseases of the Nervous System, Washington, D. C., September 9, 1954.

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The disease apparently occurs more often in males of middle age and older. The onset is insidious, and the course inevitably progressive to a fatal termination in an average of three years, but infrequently in less than one or as long as ten years. The characteristic clinical signs and symptoms are progressive weakness and atrophy, with fasciculation and fibrillation of affected muscles; most commonly these begin in the small muscles of one hand although they may begin in any of the striated musculature. The corticospinal or corticobulbar tracts may become involved at the same time in the course of the disease, giving rise to pseudobulbar symptoms, spasticity of the extremities with increased muscle stretch reflexes and extensor plantar responses but not usually with the loss of the abdominal reflexes. If muscle atrophy is severe the reflexes may be absent in spite of corticospinal-tract involvement. Sensation and intellect are almost always unaffected. The vesical and rectal sphincters are seldom affected. The usual cause of death is inanition accompanying dysphagia or aspiration pneumonia.

TABLE 3. *Mortality of Amyotrophic Lateral Sclerosis (Including Progressive Muscular Atrophy) in Selected Countries.**

COUNTRY	DATE	NO. OF DEATHS	RATE PER 100,000 POPULATION	RATIO OF MALES TO FEMALES
U. S. (including Hawaiian Islands)	1949	1007	0.7	1.8
Canada†	1951	113	0.8	1.5
Norway	1951	29	0.9	1.7
England and Wales	1952	476	1.1	1.8
Scotland	1952	50	1.0	2.1

*Based on Kurland & Mulder.[29]

†Includes certificates in which amyotrophic lateral sclerosis listed as "contributory" cause of death (accounting for about 10 per cent of total).

Epidemiologic Characteristics

Geographic distribution. Mortality statistics for amyotrophic lateral sclerosis reveal no great variation in the rates for the countries from which these statistics were available (Table 3). In the regions of the United States and Hawaii the rates are similar, although that for the South is slightly lower than those for the other regions (Table 4). These rates are for the white and nonwhite populations combined, and since the rate reported for nonwhites is lower than that for whites the combined rate in the South is

affected most. The rates for whites in all regions are about the same.

The annual mortality for amyotrophic lateral sclerosis in the countries listed thus averages just under 1 per 100,000 population (Table 3). If adjustment for possible underreporting is allowed a rate of about 1.4 per 100,000 seems reasonable. The average duration for amyotrophic lateral sclerosis from onset to death is about three years. An approximation of the prevalence rate may be made for a disease such as amyotrophic lateral sclerosis, which is practically always fatal, by multiplication of the mortality rate by

TABLE 4. *Mortality of Amyotrophic Lateral Sclerosis (Including Progressive Muscular Atrophy) in the United States, by Region, 1949.**

REGION	NO. OF DEATHS	RATE PER 100,000 POPULATION
Northeast	206	0.7
North Central	339	0.8
West	135	0.7
South	244	0.5
Territory of Hawaii	3	0.6

*Based on Kurland & Mulder.[29]

"average duration" in years. For the countries and regions reported, the prevalence ratio is therefore about 4 per 100,000 population.

The unusually high prevalence rate on the island of Guam has recently come to light through the reports of Zimmerman,[30] Arnold, Edgren and Palladino,[31] Koerner[32] and Tillema and Wijnberg.[33] Kurland and Mulder,[29] in their intensive survey in the Western Pacific islands, found the disease to be highly prevalent only among the Chamorro people of the Mariana Islands. The disease, which is clinically and pathologically characteristic of amyotrophic lateral sclerosis, has apparently been highly prevalent there for generations and perhaps for centuries. At present about 8 to 10 per cent of all adult deaths on Guam are due to this cause. After intensive surveys on Guam and Rota, the prevalence rate was estimated at about 420 per 100,000 population, or, as Arnold and his associates[31] had previously reported, at least "one hundred times greater than the estimated rate of occurrence in this country [United States]."

Population selectivity. Amyotrophic lateral sclerosis is principally a disease of middle-aged adults (Table 1). Two thirds of the cases begin between thirty-five and fifty-five years of age. In the mortality statistics reported here (Table 3) and in most clinical series, the disease is reported more often in males. The reason for the higher rate in males is unknown; some have suggested that hard labor is a predisposing factor. On the other hand, perhaps the relatively greater amount of adipose tissue in the extremities of females makes it more difficult to observe muscle fasciculations and atrophy in women and this may cause some underreporting of the disorder in females.

In the past, few reports have given any indication of some factor of etiologic significance for amyotrophic lateral sclerosis.

Brown[23a] recently described a case associated with chronic mercurialism. Alpers and Farmer[34] reported 2 cases of this disorder occurring in men who operated pneumatic drills, but since no other such occupational selectivity has been reported it seems more reasonable to accept these 2 cases as chance occurrences. The same reservation might be made for the occasional cases of progressive muscular atrophy reported in persons who had previously suffered from acute anterior poliomyelitis.[35,36]

Various inflammatory disorders such as syphilis and epidemic encephalitis are reported to have caused clinical syndromes that bear some resemblance to idiopathic amyotrophic lateral sclerosis. However, these disorders are usually readily distinguished by clinical observation with the assistance of appropriate laboratory studies. Recently, Konovalov[37] reported the possible isolation of a virus in 1 case, but there is considerable doubt that such results will be repeated by others. Inflammatory changes are occasionally found in central-nervous-system tissues of patients with no history of encephalitis but with the clinical picture[38] of amyotrophic lateral sclerosis. However, such findings are exceptional, according to Lawyer and Netsky,[28] who failed to observe any significant inflammatory changes in 53 consecutive autopsies on patients with amyotrophic lateral sclerosis.

During the last decade vitamin E has been widely used in the therapy of amyotrophic lateral sclerosis, but after repeated therapeutic failures its use has been discouraged. No clear evidence that the disease is due to a deficiency of vitamin E or any other vitamin has come to our attention.

The association of amyotrophic lateral sclerosis and acromegaly was thoroughly discussed in a case confirmed at autopsy by McCullagh and Hewlett,[39] who also described 2 cases of acromegaly in which amyotrophy developed, 1 patient improving with treatment of the acromegaly. According to Adams and his associates,[27] muscle atrophy in patients with acromegaly and Frölich's syndrome has been observed by others as well. However, Adams et al. believe that some of the cases described are "fortuitous concurrences of unrelated diseases, and others may be examples of thyrotoxic mycopathy, since hyperthyroidism not infrequently develops in acromegalic patients."

Two recent interesting reports of other associated disorders should be mentioned. Barris[40] described 5 cases of progressive muscular atrophy in patients with hyperinsulinism due to pancreatic adenoma. In 4, the atrophy was arrested after removal of the pancreatic tumor. Ask-Upmark[41] reported 5 cases of amyotrophic lateral sclerosis after gastrectomy. However, the total number of gastrectomies in which these 5

cases occurred is not specified so that it is not possible to be sure whether the rate for this group is greater than would be expected by chance.

In none of the patients observed nor in the cases reviewed by Kurland and Mulder on Guam or at the Mayo Clinic has any relation of amyotrophic lateral sclerosis with pancreatic adenoma or gastrectomy been noted.[24,29]

Genetic Characteristics

In Guam the familial prevalence of amyotrophic lateral sclerosis appears to be greater than would be expected by chance. Folklore regarding the origin of the disease indicates that members of certain families have been particularly afflicted for over a hundred years. In a southern village of Guam[9] one family history disclosed that 3 siblings, a paternal uncle and aunt and 4 paternal cousins were or had recently been affected, and it was found that in this particular family amyotrophic lateral sclerosis had been highly prevalent for generations. To date, no external etiologic factor has been detected in the investigation in the Mariana Islands.

However, according to practically all recent textbooks of neurology and clinical reviews of cases, amyotrophic lateral sclerosis is "not familial and is not hereditary."[24,27] Because the epidemiologic and genetic findings in the Mariana Islands did not agree with this concept, the question of possible genetic factors in the etiology of this disorder was reviewed. One of us (L.T.K.) had the good fortune to observe at the Mayo Clinic a patient who reported that several other members of his family had been similarly afflicted. After interviews with members of his family and a review of the medical records of his relatives, a pedigree was made up (Fig. 2), revealing 12 cases in 4 generations. Inquiries among several of the neurologists at the Mayo Clinic led to the development of 5 other pedigrees of families with hereditary amyotrophic lateral sclerosis, providing a total of 6 pedigrees with 34 cases. In 1 of these pedigrees a father and 4 of his 5 children died of the disease. In another a father and 3 of his 4 children died of amyotrophic lateral sclerosis; a brother and a niece of the father, although not examined by us, had a characteristic history, and the grandfather and a sister of the father had had neurologic abnormalities of uncertain diagnosis.

Autopsy data were obtained on 3 members of these last families. In 2 sisters of the first family chronic muscular atrophy and anterior-horn-cell degeneration were found; however, in only 1 was there demyelination in the lateral column. In the third patient there was muscle atrophy, anterior-horn-cell degeneration, some lateral-column degeneration and slight demyelination in the posterior column as well. The details of these and the other pedigrees have recently been reported elsewhere.[24]

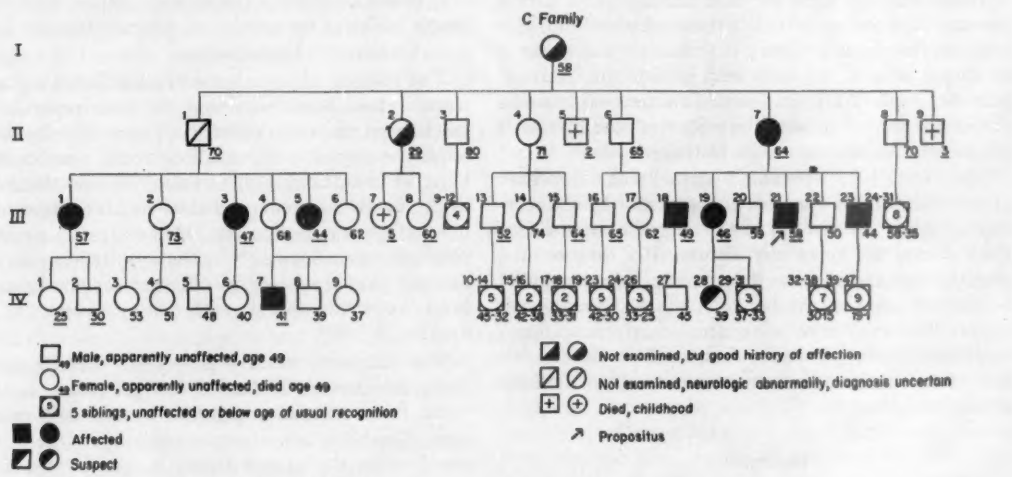

FIGURE 2. *Pedigree of C Family (Reproduced from Kurland and Mulder[24] with the Permission of the Publishers).*

On a review of the medical literature 18 pedigrees with 90 cases (not including the experience on Guam) were found. The earliest report was that of Aran[42] (1850), who described the occurrence of progressive muscular atrophy in a "45-year-old ship captain whose sister and two maternal uncles had died of a generalized muscular atrophy." The most extensive pedigree is that first reported by Osler in 1880 and more recently brought up to date by Brown.[43] There are at least 17 affected persons in 3 generations in this family.

Each of the 24 pedigrees with a total of 124 cases (67 males, 57 females) is compatible with a dominant inheritance. It was noted, however, that some persons who apparently inherit and transmit the trait may, on occasion, remain free of the disorder themselves. The

male-female ratio of the persons affected in these pedigrees is about 1.2:1. However, examination of the sex difference in the complete sibships in these pedigrees revealed no significant difference in the proportion of affected males and females.[24] The discrepancy between this finding and that reported in the mortality statistics above and in other reports has not yet been clarified.[24]

In a further review of Mayo Clinic records of 58 Minnesota patients with amyotrophic lateral sclerosis seen from 1945 to 1953 a presumptive history of other cases in the family was obtained in 5 cases. A note on the chart indicating a suspicion that the neurologic condition was hereditary was found in 2 additional records.

These findings indicate that amyotrophic lateral sclerosis does occur as a hereditary disease and it is believed that such a history is to be noted in about 5 to 10 per cent of the cases seen outside the Mariana Islands. In the Mariana Islands the best explanation at present is that a hereditary form of the disease is responsible for the very high prevalence there.

The so-called "sporadic" cases seen elsewhere greatly outnumber the hereditary cases. It is uncertain whether the etiology of the sporadic cases differs from that of the hereditary disease. If it does not the possible explanation for the relative infrequency of hereditary cases might be "inadequate" family histories, the occurrence of unrecognized spontaneous mutations or the possibility that some persons inherit and transmit the trait but remain unaffected themselves.

Discussion

The contrast of the characteristics of the two disorders described here is a strong indication that those who prefer to classify them together as "demyelinating diseases" have selected a very minor similarity. Perhaps as the etiology of one is clarified, these differences will guide investigators in their search for the etiology of the other.

The principal points of contrast here relate to the geographic distribution and genetic characteristics.

The prevalence of multiple sclerosis, at least as determined in the epidemiologic studies in North America, varies with geographic latitude but not with season or by year. Within any community or region, the illness appears to be distributed at random among all nationalities, social levels and occupational groups. It occurs in both sexes, perhaps slightly more often in females, and appears to be selective only in age of onset, since adults, mainly young adults, are affected. Contagion and pregnancy play no decisive part in its selectivity.

The role of heredity in the disorder remains uncertain. Millar and Allison, Pratt and his associates[20] and Müller[21] noted similar small ratios of affected siblings or parents to unaffected siblings or parents of the patients. Pratt et al.[20] stated that the accumulated evidence indicated that "a genetic factor is present in disseminated sclerosis." However, he went on to conclude that "the aetiological factor or factors causing disseminated sclerosis may act more readily in the presence of an inherited disposition towards this disease." Müller,[21] on the other hand, faced with similar results in his statistical analysis, concluded, "The available data gave no support to the concept that genetic factors are active in multiple sclerosis."

The data on geographic distribution indicate that an exogenous factor is probably of prime importance in the etiology of this clinical syndrome, but whether the presence of the occasional indigenous cases in the South indicates that one specific causative factor that may be responsible for the majority of the cases in the North is absent or merely less prevalent in the South is still a matter of speculation.

The relation of prevalence to some factor of "cold" climate has been suggested in this report. The mechanism of action of such a factor is unknown; it might be surmised that chilling could alter coagulability of blood and tend to cause venular thrombosis in the nervous system, and this has been suspected as one pathogenic mechanism. However, such a concept remains unconfirmed.[44,45] Dietary deficiencies and excesses that may vary in different regions have also been investigated but with little success, in our opinion.[12,17,44-46]

The similarity of the geographic distribution of multiple sclerosis to that of such disorders as rheumatic fever remains a point of interest and speculation. Disabling acute respiratory disease, at least as manifest in the armed forces in the United States during World War II,[47] also shows a higher rate of prevalence in the North than in the South. The cause of rheumatic fever is unknown, but a hypersensitive reaction to streptococci (and perhaps a virus) is suspected. It has been suggested[48,49] that multiple sclerosis may also be a manifestation of an allergic hypersensitivity. The analogy between the tissue-specific type of hypersensitivity reactions reported in various animals inoculated with kidney and heart tissue[50] and that of the demyelination produced in animals inoculated with brain substance[51] might also be cited as evidence of an "iso-allergic" factor in multiple sclerosis. However, the sequence of events that could "initiate such an allergic reaction in human beings (liberation from the central nervous system of the tissue antigens stimulating antibody formation, which in turn react with the original tissue) . . . are as yet completely unknown."[48,49]

In spite of the failure of such agents as cortisone to be of proved effectiveness in the treatment of multiple sclerosis we prefer to consider the epidemiologic and genetic data as supporting evidence that an induced hypersensitivity is a responsible factor for multiple sclerosis until a better hypothesis is proposed.

The variation of the frequency of multiple sclero-

sis with latitude has led to the recommendation[52] that patients migrate to warmer climates. The effect of such a change of residence has not been carefully evaluated since no patient group has been followed to determine the effect of change of climate. The life expectancy of patients with multiple sclerosis in different communities in North America has been compared, but the results are indecisive.[12] It seems that until a specific treatment is developed migration to a warm climate might be worth trying, but *only* if there is no severe economic or emotional hardship to the patient and *only* if he recognizes that it is uncertain that such a move will be of any benefit.

The clinical and pathological characteristics of amyotrophic lateral sclerosis have been described briefly. In contrast to the disseminated demyelinated lesions of multiple sclerosis, amyotrophic lateral sclerosis appears to be a motor-neuron or motor-system disease. The demyelination usually present in the lateral columns is probably secondary to motor-cortex-cell degeneration rather than a primary type of demyelination. The relation of the infrequently observed posterior-column demyelination to the rest of the pathological picture is not yet understood.

The geographic distribution of amyotrophic lateral sclerosis, according to the mortality statistics presented in Tables 3 and 4, shows no great variability. This suggests that the etiologic factor is evenly dispersed throughout these regions and countries. Thus, climatic factors, diet, many vector-borne infectious agents and other possible external factors can probably be discarded as etiologic agents.

The relatively high prevalence of amyotrophic lateral sclerosis among the Chamorro people of the small islands of Guam and Rota, its absence among other races living on the same islands and its occurrence among Chamorros living away from the islands suggest a hereditary factor in these people. The mechanism by which the situation in the Marianas might have arisen will be reported in the future.[53]

Recently, Kurland and Mulder[29] reported a large number of extensive pedigrees of amyotrophic lateral sclerosis and its clinical components, progressive muscular atrophy and progressive bulbar paralysis. Evidence was presented that a significant proportion of the cases of amyotrophic lateral sclerosis are due to a hereditary factor. It is uncertain whether the etiology of the sporadic cases, which still account for the large majority of all cases, differs from that of the hereditary.

In the light of the recent provocative reports on hereditary amyotrophic lateral sclerosis, it seems appropriate to present a pertinent quotation from Marks's[38] review of possible etiologic factors in amyotrophic lateral sclerosis:

> The selective involvement of both upper and lower motor neurones favors the concept of abiotrophy, a condition defined as a "premature loss of vitality or degeneration of certain cells or tissues not caused by any apparent external influence." Such an interpretation, however, sheds little light on the actual causation of the disease; moreover, the infrequency of amyotrophic lateral sclerosis in families, the rare examples of direct inheritance and the onset of symptoms relatively late in life, speak against abiotrophy as the dominant causative factor.

If points in this quotation are reviewed, one by one, in the light of the evidence presented, it will be seen that "abiotrophy" may indeed be the best lead in the search for etiology. In the first place, evidence has been presented that familial amyotrophic lateral sclerosis is not infrequent; secondly, when it does occur, direct (or dominant) inheritance is the usual pattern (although the argument in favor of abiotrophy would be equally valid if inheritance were recessive); and, thirdly, the late onset in life is not a good argument against abiotrophy since Huntington's chorea, a recognized "abiotrophy," begins at about the same age as amyotrophic lateral sclerosis. To return to Marks's quotation, it seems that his statement that "such an interpretation [that is, of abiotrophy] sheds little light on the actual causation of the disease" is incorrect, for it is better to know something of the cause than to flounder around with false leads. However, it seems desirable to reconsider the use of the term "abiotrophy." Adams and his co-workers[27] state, "The old terms abiotrophy or degeneration are now deprecated because they imply an intrinsic lack of viability or a premature senescence for which there is no evidence." Perhaps it would be more constructive to think of the abiotrophies in the sense of inherited metabolic disorders in which the nervous-system manifestations of the disease are most prominent. However, the mode of action of the gene or genes that give rise to the pathogenic process may be outside the nervous system. Hepatolenticular degeneration is still referred to as an abiotrophy in spite of the fact that evidence is available that improper copper metabolism, apparently related to liver dysfunction, results in the nervous-system disease.[54] The same point might be made for subacute combined degeneration of the spinal cord, in which abnormal metabolism in the gastric mucosa apparently leads to the nervous-system "degeneration."

So far as amyotrophic lateral sclerosis is concerned, the evidence presented supports the point of view held by others, including Adams, Denny-Brown and Pearson,[27] that amyotrophic lateral sclerosis is a metabolic disorder with a predilection for motor neurons; it is conceivable that the basic pathogenesis lies outside the nervous system. Perhaps the underlying disorder will be found associated with the upper gastrointestinal tract. Until some associated anatomic or physiologic abnormality is observed, however, this remains a speculative matter. The discovery of such an association should lead to a better understanding of the pathogenesis of amyotrophic lateral sclerosis and to therapeutic success instead of its present deadliness.

Summary and Conclusions

Recent epidemiologic and genetic data in association with clinical and pathological observations are compared for multiple sclerosis and amyotrophic lateral sclerosis. The most striking epidemiologic characteristic for multiple sclerosis is its rarity in the tropics and subtropics and its relatively high prevalence in regions with a "colder" climate. No selectivity for race or nationality within any particular area was observed. The importance of a genetic factor in multiple sclerosis is equivocal, and the data strongly suggest that an exogenous factor is of prime importance.

On the other hand the geographic distribution of amyotrophic lateral sclerosis does not vary in the countries and regions studied except for the Mariana Islands, where this illness is exceedingly prevalent. No external etiologic factor has been discovered on Guam in the Mariana Islands or elsewhere, but the occurrence of numerous cases in some families and none in neighboring families was noted. In a recent genetic study of amyotrophic lateral sclerosis, a large number of pedigrees indicative of dominant inheritance were collected. It is uncertain whether the "sporadic" cases of amyotrophic lateral sclerosis that still account for the majority of all cases observed in practice have a basic etiology differing from that of the hereditary form of the disease. The data strongly suggest that for amyotrophic lateral sclerosis exogenous factors are of little importance etiologically; it is inferred that this disorder is due to some yet undetected metabolic disturbance that develops in adulthood and is often inherited.

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UNUSUAL MANIFESTATIONS OF CARCINOMA OF THE ESOPHAGUS*

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PHILADELPHIA

Approximately 20 per cent of esophageal carcinomas are nonobstructing when first seen and many remain so throughout the course of the disease.[1-4] For this reason dysphagia, usually the most prominent symptom, may be a late or terminal manifestation.[1,5,6] In the nonobstructing lesions unusual presenting symptoms often occur as a result of extension to contiguous structures or as a consequence of distant metastasis. In either event the patient's complaints may seem unrelated to the esophagus, and the correct diagnosis may be missed or delayed.

Local extension can cause hoarseness, pain or cough from involvement of the recurrent laryngeal nerve or tracheobronchial tree. As infiltration of structures adjacent to the esophagus continues, fistulas may develop, with resultant mediastinal abscess, pneumonia or lung abscess that lead the patient to seek medical care. Caudal metastasis to abdominal lymph nodes or viscera may result in gastrointestinal symptoms simulating gastric carcinoma, peptic ulcer or other intra-abdominal disease.

Franklin and Shipman[7] reported 2 cases of carci-

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noma of the cervical esophagus in which throat symptoms led to erroneous diagnoses. The first patient was thought to have angioneurotic edema, which temporarily responded to antihistamine therapy. Later dysphagia developed, and on study an esophageal carcinoma was found. The second patient had a tonsillectomy for throat pain, and dysphagia subsequently developed, leading to the correct diagnosis of carcinoma of the esophagus.

Case Reports

Case 1. C.R., a 57-year-old man, was admitted to the hospital with a history of a change in the tone and character of his voice to a rasping quality 1 year previously. Six months later he began to have increasing difficulty in swallowing solid food.

On admission right-vocal-cord paralysis was seen on direct laryngoscopy. X-ray study was characteristic of nonobstructing carcinoma. Esophagoscopy revealed a nodular lesion of the thoracic esophagus that proved on biopsy to be a moderately differentiated squamous-cell carcinoma. The patient did not survive partial esophagectomy and esophagogastrostomy.

This patient was hoarse at least six months before the onset of dysphagia. Appropriate studies might well have revealed the diagnosis long before the development of this symptom.

Case 2. F.F., a 75-year-old man, was first seen on February 20, 1954, with a history of cough of approximately 2 months' duration. The cough, productive of small amounts of white sputum, was present intermittently during the day and more severely at night. He complained of anorexia, but no dysphagia, nausea or vomiting. There were no other symptoms, and he had always been in good health.

X-ray examination of the chest showed a small zone of consolidation in the right middle lobe that was considered most probably due to a resolving segmental pneumonitis. He refused any further studies although he continued to have severe cough, only partially relieved by expectorants. It was elicited at this time that although solid food produced no discomfort coughing was often induced by swallowing of liquids, particularly hot ones. On July 5, because of progressive increase in the severity of the cough, he was hospitalized. As part of a complete diagnostic survey x-ray examination of the esophagus revealed a lesion in the middle third nearly 8 cm. in length, with ulceration in juxtaposition to the lower end of the trachea and the left main bronchus. There was no evidence of obstruction and no dilatation above the lesion. On esophagoscopy a large ulcerating lesion encircling the esophagus was seen. Biopsy disclosed squamous-cell carcinoma. There were still no obstructive symptoms. Because of the proximity of the lesion to the trachea moderate dosage of 250-K.V. rotational therapy was planned with the intention of giving 3000 r to the lesion. After a total dosage of approximately 900 r (tissue dose) an esophagotracheal fistula developed, and treatment was discontinued.

This patient had cough for seven months, probably resulting from the proximity of the ulcerative lesion to the trachea and left main bronchus. At no time were there symptoms of obstruction despite the presence of a large esophageal lesion. Even after the development of a tracheoesophageal fistula under therapy cough remained the only prominent symptom.

Case 3. A.T., a 54-year-old man, was admitted to the hospital with a history of anorexia of 8 months' duration. For only 2 or 3 weeks had he had dysphagia, cough after swallowing and vomiting immediately after eating. He had lost a great deal of weight. On admission he had chills and fever and stated that swallowing of food caused severe cough, pain in the right side of the chest and expectoration. Examination of the chest revealed dullness, decreased breath sounds and moist rales over the right lower lobe. X-ray study showed consolidation of the right lower lobe, with a large abscess cavity containing a fluid level. This was thought to be a lung abscess secondary to cavitating pneumonia or a pulmonary neoplasm. On administration of barium for a gastrointestinal series the cavity in the right lower lobe filled with barium even before the stomach. There was an irregularity in the lower third of the esophagus, with a sharp shelving defect at the junction of the middle and lower third. Esophagoscopy confirmed the presence of an infiltrating tumor extending around the circumference of the esophageal wall. The biopsy diagnosis was squamous-cell carcinoma.

Although the known duration of symptoms was eight months, dysphagia had been present for only two weeks. The prominent symptoms even at this time were those related to pulmonary suppuration secondary to a tracheoesophageal fistula.

Case 4. M. McG., a 51-year-old man, had a dry, continuous cough and chest pain in March, 1954. He was treated at home for "pneumonia." After this he became anorectic, lost 22 pounds and continued to cough, at first with blood-streaked sputum and later with gross hemoptysis. Studies for tuberculosis were negative. On July 9 he was admitted to a hospital, where stereoscopic x-ray examination of the chest revealed a homogeneous density in the left apex. Bronchoscopic examination showed stenosis of the left-main-stem bronchus, compression of the left-upper-lobe bronchus and purulent secretion behind the compression. There was no ulceration, and no tissue was obtained for biopsy. At exploratory thoracotomy a diagnosis of carcinoma of the lung, with metastases to mediastinal lymph nodes, was made. No biopsy was taken. The patient was discharged from the hospital and because of progressive weakness, nausea and vomiting was admitted to the Philadelphia Veterans Administration Hospital on September 15. X-ray examination revealed a paramediastinal mass on the right. The nature of the lesion could not further be defined by laminagraphy or pneumomediastinum. Although x-ray study of the esophagus was made the changes were so minor that they were not recognized at this time. The nausea and vomiting were relieved by chlorpromazine. Despite failure to obtain a positive tissue diagnosis of neoplastic disease x-ray therapy was instituted on the basis of the findings on bronchoscopy and at thoracotomy. The patient went home on November 23 and was readmitted to the hospital January 17, 1955, because of progressively increasing chest pain, weight loss and some difficulty in swallowing. After several days the dysphagia was relieved except for a burning sensation in the lower substernal region when solids were ingested. X-ray study of the esophagus revealed a definite stenosing deformity. Esophagoscopy was suggested to determine if the lesion was malignant but could not be carried out because of the patient's condition. He died on January 28. At autopsy the lesion proved to be a carcinoma of the lower third of the esophagus. There was no invasion of the lung although the mediastinal lymph nodes were involved.

The late development of dysphagia in this case and the unusual presenting symptoms were responsible for failure to diagnose the lesion even at exploratory thoracotomy.

Case 5. M.C., a 43-year-old man, was admitted to the hospital for hematemesis of 2 days' duration. Ten days previously he had first noted epigastric pain relieved by food. That evening he choked and vomited, and after this on several occasions he vomited bright-red blood and had tarry stools. He also complained of pain in the epigastrium and left aspect of the hypochondrium.

The initial diagnosis was either bleeding peptic ulcer or gastric carcinoma. A gastrointestinal series showed an intrinsic defect on the lesser curvature of the stomach, with

extravasation of barium into the lesser peritoneal cavity. At operation there was a large neoplastic mass in the stomach, with extensive abdominal metastases in addition to direct extension to the liver. A biopsy revealed the lesion to be metastatic squamous-cell carcinoma. At autopsy 3 months later the primary lesion proved to be a carcinoma, 3 cm. long, in the middle third of the esophagus.

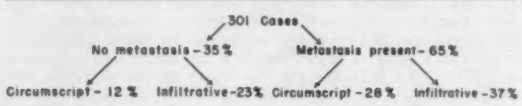
TABLE 1. *Frequency of Metastases Found at Autopsy in 192 of 301 Patients.**

SITE	NO. OF PATIENTS
Lymph node	153
Liver	62
Lymphatic channels in esophagus	37
Lungs	20
Kidney	19
Bone	10
Pleura	8
Adrenal gland	8
Thyroid gland	6
Myocardium	5
Omentum	5
Pancreas	4
Spleen	3
Peritoneum	3
Intestine	3
Pericardium	3
Stomach	2
Tongue	1
Epiglottis	1

*Based on data of Neuberger.[8]

The symptoms in this case, which were indeed of brief duration, were not related to the primary tumor but to metastatic involvement of the stomach and other abdominal organs.

TABLE 2. *Autopsy Study of 301 Cases of Carcinoma of the Esophagus.**



*Based on data of Neuberger.[8]

Symptoms resulting from distant metastasis are even more difficult to relate to a primary esophageal lesion. The prevalence and site of metastasis from carcinoma of the esophagus has been studied by Neuberger[8] and suggests the frequency and distribution with which unusual presenting manifestations may be expected in nonobstructing lesions (Tables 1 and 2). The rich lymphatic drainage of the esophagus and the many intercommunicating channels explains the widespread metastases that may occur (Fig. 1).[9] Metastatic lesions in regional or distant lymph nodes may simulate primary neoplasms of other organs. In metastatic carcinoma, wherever the lesions are found the esophagus must be considered a possible primary site.

CASE 6. G.H., a 47-year-old man, was admitted to the hospital with a 3-month history of pain in the right side of the neck. Two months before admission hoarseness and a productive cough developed. Several weeks later a mass in the right side of the neck was noted, and the pain radiated from the neck down the ulnar aspect of the right arm. There had been a weight loss of 8.2 kg. (18 pounds). At no time was there any dysphagia.

Examination revealed a large, firm, nontender, lobulated mass, approximately 6 cm. in diameter, at the base of the neck in the region of the right lobe of the thyroid gland, extending into the right supraclavicular fossa. The mass moved with deglutition. There were also several small, firm lymph nodes in the right anterior cervical chain. Right-vocal-cord paralysis was present. A clinical diagnosis of carcinoma of the thyroid gland was made.

X-ray study of the chest demonstrated numerous nodular densities of varying sizes distributed diffusely throughout both lungs. These were characteristic of pulmonary metastases. The greater number of metastatic foci in the lung bases was considered suggestive of metastatic thyroid carcinoma. No mediastinal widening was present. Bone survey was negative.

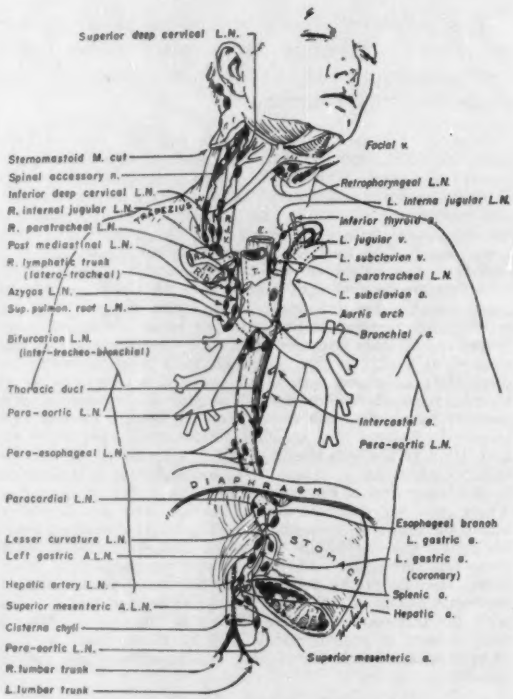


FIGURE 1. *Lymphatic Drainage of the Esophagus.*

Since no uptake of radioactive iodine was demonstrated in either the neck mass or the chest metastases, total thyroidectomy was planned in the hope of inducing enough compensatory activity in the metastases to permit later treatment with radioactive iodine.

At operation a large white tumor mass attached to the thyroid gland but not infiltrating it was found. Microscopical examination of the tissue revealed squamous-cell car-

cinoma. A subsequent esophagram demonstrated a typical nonobstructing infiltrating annular carcinoma of the esophagus at the level of the aortic arch. This diagnosis was confirmed at autopsy 6 weeks later.

Metastases to the thyroid gland from a nonobstructing esophageal carcinoma led to an erroneous diagnosis of thyroid carcinoma. Only biopsy of the lesion and a search for a primary tumor resulted in a correct diagnosis.

CASE 7. J. L., a 47-year-old man, first noticed a mass in the right side of the neck 6 weeks before admission that gradually increased in size until it was about 6 cm. in diameter. There was no history of pain, weight loss, cough, hoarseness or dysphagia.

Physical examination showed a soft, fluctuant, spherical mass in the right supraclavicular fossa. The skin over it was taut. A satellite group of hard, fixed lymph nodes attached to underlying structures was present. There was no evidence of inflammation. The thyroid gland was not enlarged. The left supraclavicular region was normal. X-ray study of the chest demonstrated numerous tiny nodules that through growth in serial studies were proved to be pulmonary metastases. Shortly after admission paralysis of the right vocal cord developed. Skin breakdown and rapid exophytic growth of the tumor occurred.

Biopsy of the neck mass showed squamous-cell carcinoma. In the search for a primary focus, both an x-ray study of the esophagus and an esophagoscopy were performed. A nonconstricting fungating lesion in the midthoracic esophagus was found, measuring approximately 4 cm. in length. The diagnosis of carcinoma of the esophagus was confirmed at autopsy 8 months later.

A solitary supraclavicular tumor was the only symptom of a large nonobstructing esophageal carcinoma. Only after biopsy of the tumor mass and a search for a primary site was the diagnosis established.

DISCUSSION

Because of the occurrence of metastases to bone, esophageal carcinoma should be considered in the differential diagnosis of bone tumors. Thompson[10] saw an elderly woman with back pain due to an osteolytic lesion of a thoracic vertebra. At autopsy an unsuspected primary carcinoma of the esophagus was found. Franklin and Shipman[7] reported 2 similar cases. The first patient had an amputation for a tumor of the tibia after a biopsy diagnosis of fibrosarcoma; study of the specimen revealed squamous-cell carcinoma, and a search for a primary site disclosed an esophageal carcinoma. The second patient had a lytic tumor of the mandible. A biopsy specimen was erroneously diagnosed as adamantinoma. The lesion later proved to be a metastasis from a primary esophageal carcinoma.

With the increasing availability of definitive surgery and radiotherapy for the palliation and treatment of esophageal carcinoma, a lesion that accounts for 2 per cent of all deaths from cancer, early diagnosis is essential.[3,11] In many cases this requires an awareness of the varied guises in which the nonobstructing lesion may present itself to the physician. Of 16 patients with carcinoma of the esophagus observed over a two-year period, 6 (42 per cent) had unusual presenting symptoms that did not suggest an esophageal lesion. When it is suspected the lesion can be diagnosed by the radiologist with reasonable certainty in 75 per cent of cases. In an additional 15 per cent he can find suggestive evidence so that esophagoscopy for confirmation will be done.[5] Because of the remaining 10 per cent in which x-ray changes are not present sufficiently early to be recognized by the radiologist a negative esophagram does not obviate the need for esophagoscopy in the search for esophageal carcinoma.

CONCLUSIONS

The diagnosis of nonobstructing carcinoma is often delayed or missed because of the absence of dysphagia. Unusual presenting symptoms may occur as a result of extension to contiguous structures or as a result of distant metastases.

The early recognition of nonobstructing esophageal carcinoma depends on an awareness of this problem and a careful search for the lesion by x-ray study and esophagoscopy.

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SPECIAL ARTICLE

FLYING DOCTORS OF THE AUSTRALIAN OUTBACK

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IT is difficult to imagine an efficient medical service functioning over the whole of a 3,000,000-square-mile country with a population of only 8,500,000 people (an estimated 2.9 persons per square mile in 1952). That is an area as large as the United States; most of the people live in 5 state-capital cities and a number of smaller towns confined almost wholly to the eastern end of the country. Some of the cities have standards of living and culture as high as those in the United States, but the center of Australia, perhaps four fifths of the country equaling all the United States except part of California and the dozen eastern states clinging to the coast, is inhabited by farming families and small communities with 50 to 1000 miles between them.

This is because in the drier areas 10 to 50 acres of land are needed to support each animal, and so farms range in size from 10,000 acres to 10,000 square miles. I encountered 1,000,000-acre farms more than once while journeying across the interior of the continent. The greater part of the country is more or less arid, and most of the lakes and rivers marked on a map are quite dry except after a sudden but infrequent rain. Wet or dry the country is difficult to negotiate by American standards. In 1947 I visited a small town in the southeastern part of Australia that I was given to understand had not seen rain for nine years. Three months later I was back again, bogged. There had been rain between my visits, and I was in mud to my axles. When it was dry I was bogged too, but that was in dust.

In spite of the vast distances and the difficult terrain, no one in Australia is too isolated to receive prompt treatment in sickness or injury, or to be removed to a hospital at short notice. And the service is both efficient and inexpensive.

The Australian Flying Doctor Service took many years of idealistic planning before it became an accomplished fact. It demanded close co-operation and the exploration of entirely new ideas by physicians, radio and aeronautic experts and other men of vision prepared to face danger without reward to help the pioneers of their country's outback areas, to whom doctors sometimes had to struggle for many days through deadly country only to arrive too late to be of help.

Australia's population was only a little more than 6,000,000 when the service began in May, 1928, under the inspiring leadership of the Rev. John Flynn, whose epitaph reads "He brought gladness and rejoicing to the wilderness and solitary places." And in spite of the countless hazards the service has never failed since then. I know these hazards at first hand. I have crossed Australia twice from one side to the other, nearly 3000 miles each way, and with almost two thirds of it across desert country, without proper roads, with hardly any water, and with little possibility of finding supplies or gasoline. Everything had to be carried, and American automobile tires sometimes gave me no more than 4000 miles before I had to discard them.

John Flynn's dream of a medical service for everyone in this rugged outback land seemed so fantastic in the 1920s that he was considered to be nothing more than a harmless visionary. But his faith and unquenchable enthusiasm achieved what money could never have achieved alone: a co-ordination of will and effort by hundreds of people who could put other men's needs before personal gain.

It has always been said of Australia, "It's heaven for men and dogs, but hell for women and horses." Flynn believed that a country unfit for women was also unfit for men, and he was willing to do something about it. I've seen a handful of Flying Doctors kill with finality the tradition that the country was "hell for women," and now the women of the outback even have their babies in the bush without risk or fear. An expression of the success of Australian medical service is found in the fact that New Zealand is now the only country with a lower infant death rate than Australia. And in 1945, a war year, the over-all mortality in Australia was only 9.5 per 1000. By 1952 this had been reduced to 9.4.

GEOGRAPHIC CONSIDERATIONS

Some idea of the geography of the service can be obtained if the cities and towns of Australia (Fig. 1) are related to those in approximately similar positions in the United States. Perth (population, 317,000), the only large city in all the western two thirds of the country, coincides perhaps with Los Angeles. Darwin (population, 1000 before World War II), the most northerly port, demolished by Japanese bombing just before the Battle of the Coral Sea and now rebuilt, would probably relate to Winnipeg in Canada. Brisbane (population, 453,000), the most northerly large city in the east, would coincide with New York City for location.

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Sydney (population, 1,600,000), a few hundred miles south of it, would be about where Norfolk, Virginia, is. Canberra (population, 26,000), the federal capital, would then be somewhere near Durham, North Carolina. Melbourne (population, 1,350,000) would coincide with Jacksonville, Florida, and Adelaide (population, 442,000) with New Orleans.

None of the Flying Doctor Centers are located in these cities; instead, all are situated more inland and in the less populated towns, from which access to the larger cities is difficult. There are 7 centers, 6 in a circle and 1 exactly in the middle of the country, and all many hundreds of miles from the more populous areas where normal facilities are available. They are located in about the following positions: Wyndham would probably coincide with Bismarck, North Dakota; Port Hedland with Portland, Oregon; Kalgoorlie with Grand Canyon, Arizona; Broken Hill with Knoxville, Tennessee; Charleville with Pittsburgh, Pennsylvania; Cloncurry with Saginaw, Michigan; and, right in the dead heart of the country, Alice Springs, approximating Kansas City. Each of these centers covers many thousands of miles of unspeakably desolate country, but country that many proud and persevering people cling to and improve and even think the finest in the world. Cloncurry, the first receiving station, initially served an area of 250,000 square miles.

Activities of the Service

In recent years, flying dentists and flying nurses have been added to the service, but originally the scheme included only physicians and surgeons. There are small independent units, of course, and occasionally I carried a "trailer" eye service to a few people in the less unpopulated but still unserved parts of the country, and I found it a very satisfying experience. The year before I left Australia I planned a 10,000-mile trip into the outback with this consulting-room trailer, but it rained with a persistence and ferocity that made me realize that too frequently, in that country, the Flying Doctor is the only man who can get through. By the time the tracks had dried from quagmire to their normal condition of fine dust, it was too late to go. However, it is hoped, and with every justification, that ultimately no service will be beyond reach of the most isolated settlers.

No scheme of this kind was possible, except to a very limited degree, before the advent of radio. And even then considerable research was essential to produce cheap transmitters that could be used where no electric power was available and would be trouble-free, simple to operate and capable of carrying conversation hundreds of miles over some of the most static-ridden country it has ever been my misfortune to encounter. A "baby" transmitter was eventually designed that derived its power from a small dynamo driven by bicycle foot pedals, and although the design has since been improved, the principle has been retained because of its reliability.

The planes and especially their pilots have to be a little unusual too. They must be able to land and take off loaded from any kind of terrain, sometimes in alarmingly short distances, and in all weathers. They must carry personnel, surgical facilities and supplies, and be able to land right beside any house or shack by day or night, with never a landing strip available unless a farmer makes one himself, which, with all the land available he sometimes decides to

FIGURE 1. *Main Centers of Australia (Normally Populated Areas Are Shaded).*

do. Sometimes at night herds of terrified kangaroos leap before them and add to the hazards of landing in the light of headlamps. There are no radio beams to fly by in storms, and the pilots fly, as they say, "By guess and by God."

Not everyone in the outback owns a radio transmitter, of course; in 1947 there were 327 in use. But every homestead is connected by a single telephone wire that ultimately reaches someone with a transmitter, so that no one is really isolated. These telephone wires have saved countless lives in another unexpected way. Like many another man in unfamiliar territory, I traveled beside them when crossing the heart of the country, and often I heard how a man taken sick or injured at a distance from a homestead has contrived to break the wire, either himself or through an aboriginal. This has brought out the owner of the nearest homestead to find and repair the break in his only link with civilization; the sick or injured man has been found, and aid summoned over the wire. In addition to this means of summoning help, all Australian Inland Mission padres carry portable transceivers so that they can report any accidents or sickness they encounter.

A woman need never worry about having a baby

if she lives in the bush. She can either summon the Flying Doctor and Nurse to deliver it or, if necessary, be flown to the nearest base hospital. On two occasions I pulled into a clearing by a farm shack 80 miles east of the Western Australian border, where the farmer's wife had been delivered of 5 babies without the slightest risk or inconvenience. This shack was 86 miles from the nearest habitation, 200 from the next and 500 from Kalgoorlie, the nearest Flying Doctor center. And the farmer had to carry their meager supply of water 4½ miles each day, pulling it up from an underground lake 200 feet below ground. A farm with 90 miles between the front gate and the house (Fig. 2) is a common experience to the Flying Doctors.

A personal experience of the heat, the inexorable

FIGURE 2. *Outback Farm, with 90 Miles between the Front Gate and the House.*

and unending dust, the countless flies and other pests that such a flat, windswept terrain produces, the dust storms, the bush fires and the rare but sudden floods is the only way one can comprehend the odds against which these people fight. Sometimes, it must be an experience bordering on the delights of a dream vacation to be whisked away into a clean cool hospital where everyone is efficient and confident.

One of the reasons the Flying Doctor Service has received the wholehearted support of every Australian is its complete lack of discrimination. Whatever a man's creed, wherever he lives, and if he is rich or poor, black or white, can or cannot pay, he is treated without favors. Even the wounded in tribal disputes are treated like all other men, and they are entirely without means to pay for the service they receive. Another factor contributing to the immediate acceptance of the scheme was that it began with neither governmental nor private sponsorship in the usually accepted sense of those terms. The average Australian-born man is suspicious alike of governmental control and capitalism. He likes to avoid both if possible, or else to combine them; so he harbored little suspicion of the project started by Dr. Flynn, who was with the Australian Inland Mission formed by the Home Mission Committee of the Presbyterian General Assembly.

The nursing homes originally established by the church in isolated places could almost be said to have been John Flynn's inspiration for the medical service, which now reaches everyone who needs it. When he saw what these homes did for the few people who could reach them, he knew that their services should be at the disposal of every settler. But the cost, in money and personnel, of the homes, no matter how small and in every place they were needed, would have been impossible to meet. The obvious alternative had to be a sure means of transporting a doctor to everyone or lifting anyone from anywhere and taking him to a hospital. At the same time care had to be exercised that no established physician would be adversely affected by the scheme; accordingly, it is an unwritten law that the service never approaches to within 100 miles of a practicing doctor who has an ambulance service at his disposal.

After a few years' growth it became obvious that the service was a responsibility too great for any one religious organization to carry. In early 1933 a group of 50 responsible citizens in Melbourne formed the Victorian section of the Aerial Medical Service of Australia. Cloncurry, the original base in Queensland, carried on for eleven years without an air accident, and then in 1939 the Queensland section of the service was formed.

The Flying Doctor Service is, of course, expensive to run, especially because the average patient could never pay for the cost of air transportation and its attendant hospitalization. But, although Australians predominantly favor national health insurance, they prefer to support this particular scheme by a number of voluntary activities and levies such as a contribution on every head of sheep a man has on his station, picnics, horse races, dances and fetes. In 1944 the Service received a federal donation of £7,500 (about $25,000 at that time). But contributions have to be progressively larger to keep the scheme out of debt. Undoubtedly, the whole service will eventually join the expanding coverage provided by national health insurance that the Government is slowly introducing to the people.

The annual mileage covered by the Flying Doctor Service is necessarily high, although the wide dispersion of patients does not necessitate a great many flights per man. The year ending June, 1945, is the last year for which I can trace figures. In that year the centers flew approximately 195,000 miles, and trips averaged 650 miles each. When one considers that the doctor at Broken Hill has a territory of 280,000 square miles to serve, this low average is surprising.

I have twice been to Ceduna, a small community on the south coast in the Great Australian Bight, picking my way over rain channels and through salt spray and whirling dust, wondering what attracts people to living where the desert joins the sea. Whatever it is, there is an unusual spirit of brotherhood in these lonely outback places. Ceduna boasts its own aerial medical service operated by the Bush Church Aid Society of Australia and Tasmania. In 1945 this small organization flew 15,220 miles. But the greatest record probably goes to a lone doctor based on Forbes, New South Wales, who although not attached to any recognized service operates three personal planes and in 1945 flew 54,000 miles and lifted 46 patients for hospitalization. Included, in this mileage was a weekly schedule to eight outback communities with which he maintained a 99 per cent regularity.

Discussion

The Australian Flying Doctor Service operates with the highest possible traditions and standards of conduct to which the medical world has ever risen — one that often demands considerable self-sacrifice and offers little reward but that undoubtedly produces the greatest personal satisfaction. The badge of the Service is shown in Figure 3. Men who have signed to serve in the scheme for six or twelve months have often stayed for many years. It undoubtedly shares the sentiments of its founder, about whom Ruth Ann Curtis wrote as follows in the Australian geographic magazine *Walkabout*, as recently as 1947:

> John Flynn is a simple man. He is no fiery orator who plays on people's emotions and drags them to a false enthusiasm of which they are slightly ashamed the next day. When he sells them on an idea, their heads as well as their hearts are concerned and they stay sold. He is a quiet man with a sense of humour and a gentle chuckle. In those old days of his wide patrols through the Inland he could tell a story as well as the next man. Better, he could enjoy another man's story even more. People recognized his sincerity and confided their secret troubles to him. At the same time they brought him clocks to mend, egg-beaters to repair. He baptised, married, and buried them; took them magazines, books, fashion papers. He pulled aching teeth, sat with sick children, wiped the dishes for tired women and prayed with dying men. Always in his mind was the great need for safety, of having a doctor within call, and of being able to contact other people to alleviate the terrible aloneness which faced so many hundreds of men and women on far stations. . . .

Figure 3. *Badge of the Flying Doctor Service.*

MEDICAL PROGRESS

ENDOSCOPY*

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THE word endoscope comes from the Greek *ενδον* (within) combined with *σκοπειν* (to examine) and means an instrument for the examination of the interior of a hollow viscus. Although at first thought endoscopy might seem to cover a very narrow area, it is not so in fact, for the endoscopist sees many of the most interesting cases in the hospital, and his field of endeavor covers much important literature.

Bronchoscopy

Technic

Wetrich[1] has demonstrated an apparatus to protect the physician during the application of topical anesthesia in the tuberculous patient. It consists of a face shield to which an ordinary hand mirror is riveted.

Davis, Kretchmer and Bryce-Smith[2] have called attention to the value of bronchoscopy during tracheotomy. Not only does the bronchoscope provide an airway during the procedure but also, particularly in children, a lighted bronchoscope can indicate the point at which the trachea should be entered.

Bronchial Obstruction

Peck and Levin[3] have studied the physiopathology of atelectasis. Three major requisites are necessary for the development of atelectasis in a lung, lobe or segment. The first is that some mechanical cause of obstruction must be present. Secondly, there must be sufficient circulation to allow absorption of the air distal to the obstruction. Thirdly, there must be no

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accessory means for air entry into the distal portion of the obstructed area. These authors have described the use of trypsin by endobronchial instillation to loosen the viscid secretions of patients with atelectasis. Two cases are presented illustrating the successful use of this material in relieving chronic atelectasis due to bronchial and bronchiolar obstruction in patients with bulbar poliomyelitis.

Gans[4] performed bronchoscopic aspiration under general anesthesia in 50 consecutive children with atelectasis. In nontuberculous patients bronchoscopic aspiration led to reventilation of the collapsed lobes in 83 per cent of cases within a month. Of the 41 collapsed lobes of these patients, 16 were due to whooping cough, 11 to pneumonia, 5 to measles, 3 to massive collapse associated with empyema, and 3 to atelectasis in bronchiectatic lobes; 2 were congenital, and 1 was due to a persistent antral infection. There were no postoperative complications. Bronchoscopic aspiration in cases of atelectasis in children frequently leads to re-expansion of the collapsed lobes and therefore prevents bronchiectasis. In 13 of the 21 children with tuberculous collapse an enlarged gland was observed to be pressing on one or more bronchial openings.

Potts, Holinger and Rosenblum[5] have reported a case of an anomalous left pulmonary artery causing obstruction to the right main bronchus. They believe that this case is unique since a review of the literature has failed to reveal a similar case of dyspnea and cyanosis caused by an abnormally placed pulmonary artery. Bronchoscopic examination revealed a normal, patent larynx and deviation of the trachea to the left. The carina was sharp, and the left main bronchus was normal. However, the lower trachea and the right main bronchus immediately above the upper-lobe orifice were compressed to a small slit by pressure on the lateral wall. Surgical repair was successful, and the postoperative course was uneventful. The embryologic mishap that produced this deformity could not be explained.

Nonspecific Infectious Diseases

In a discussion of fibrinous bronchitis, Williams[6] reports that bronchial casts form in a bewildering variety of thoracic and constitutional diseases. Characteristically, the patient has repeated bouts of severe paroxysmal dyspnea and cough that terminate in the expectoration of a bronchial cast. Tuberculous bronchitis is common. At least 20 cases of fibrinous bronchitis in pulmonary tuberculosis have been recorded. Bronchoscopy should occupy an important place in the diagnosis and treatment of this rare disease, which may be rapidly fatal.

Croxatto and Lanar[7] have performed an experimental study on the pathogenesis of bronchiectasis. They report that complete bronchial obstruction leads to bronchoalveolar apneumatosis. The secreted mucus, not finding an external outlet, dilates the bronchus (mucus bronchodilatation). The mucus bronchodilatation constitutes the first phase of every obstructive bronchiectasis. The mucus bronchodilatations may, once the bronchus becomes recanalized, disappear at the beginning or become permanent later (bronchiectasis) as a result of the bronchiolar obstruction or as a consequence of an accompanying infection. It is probable that all bronchiectasis, acquired, primary or secondary, is caused by a bronchial obstruction.

Fox, Hughes and Sutliff[8] have reported their experience with 55 consecutive cases of lung abscess. They believe that bronchoscopy has been of value in ruling out tumors, foreign bodies and other intrabronchial lesions; all but a few of these patients were examined with the bronchoscope. Except for temporary aspiration of purulent material they did not see any therapeutic value in bronchoscopy. However, in a case in which segmental bronchial constriction was present, bronchial dilatation at intervals was of help in effecting a cure. It was their opinion, after reviewing the resected material, that no case not cured by antibiotic treatment would have been cured by bronchoscopic aspiration and antibiotic instillation.

Tracheobronchial Tuberculosis

Taronna and Zucconi[9] made a statistical and clinical study of bronchoscopy and primary tuberculosis in an attempt to determine the relation between tuberculosis and the bronchial tree — namely, eventual bronchial alterations induced by the presence of enlarged lymph nodes and the effects of these alterations on the respiratory function. The roentgenograms revealed adenopathy and parenchymal shadows in 16 patients, and adenopathy without parenchymal shadows in 26. Bronchoscopy showed that the primary lesions were accompanied by severe alterations of the bronchial tree in most of the first 16 patients. These bronchoscopic findings indicate that often the parenchymal shadow of the roentgenograms of patients with primary tuberculosis is due not to an allergic reaction but to hypoventilation caused by endobronchial or extrabronchial stenosis of the bronchus in the involved area. The authors consider bronchoscopy in patients with primary tuberculosis important not only from the diagnostic but also from the therapeutic standpoint.

Vaksvik,[10] in his study of primary bronchial tuberculosis in adult patients in a sanatorium, reports that true primary bronchial tuberculosis with a primary focus in the bronchial mucosa is extremely rare and of theoretic importance only. In so-called "primary" bronchial tuberculosis, the erosion of a caseous lymph node into a bronchus is the essential feature. At Glittre Sanatorium, Norway, during 1262 bronchoscopies in 1003 adult patients, 20 (2 per cent) were found to have definite lymph-node perforation into the tracheobronchial tree. The majority of these

patients had large, craterlike perforations from which pus and caseous material could be expressed.

Olson, Jones and Angevine[11] made a special study of bronchial disease in lungs resected for pulmonary tuberculosis. Six hundred and two surgical specimens resected from patients with pulmonary tuberculosis were studied with particular reference to their bronchial lesion. In only 10 per cent of the specimens were the bronchi normal. The frequency of different types of bronchial involvement was as follows: chronic bronchitis, 29 per cent; tuberculous bronchitis, 20 per cent; bronchiectasis without endobronchial tuberculosis, 21 per cent; and bronchiectasis with endobronchial tuberculosis, 20 per cent. Some degree of endobronchial tuberculosis was present in 40 per cent, and ectatic bronchi in 42 per cent, of the total specimens. It should be emphasized that 37 per cent of the specimens from patients who had negative bronchoscopic examinations actually showed endobronchial lesions. In some cases these lesions were in the submucosa of the main-stem bronchi.

Roll[12] has made a study of ulceration of the bronchial stump after resection for tuberculosis. The ulceration was considered to be tuberculosis in more than 24 per cent of 400 resections for pulmonary tuberculosis. On statistical analysis it was found that stump ulceration was significantly more frequent in the group of patients who had the highest prevalence of strains of tubercle bacilli resistant to drugs at the time of surgery. There were also more ulcers in surgical patients who had nonspecific inflammatory lesions of the bronchi. All ulcers followed satisfactorily eventually healed, usually within four months, but 1 persisted for eleven and a half months.

Belgrano[13] calls attention to the importance of stenosing bronchial tuberculosis complicating pulmonary tuberculosis in the production of irreversible bronchopulmonary syndromes of obstruction that require surgical therapy. Partial or total resection is indicated in patients with pulmonary tuberculosis and chronic stenosis of the main or lobar bronchi.

Blastomycosis

In a collective review of North American blastomycosis, Kunkel et al.[14] studied 103 cases. Bronchial inflammation, present in every case, was manifested mainly by diffuse infiltration with polymorphonuclear leukocytes and lymphocytes. In places, this reaction had caused ulceration of the mucosa. Organisms, as well as giant cells, were seen occasionally within the lumens. Reports of bronchoscopic biopsies were available in 3 of the 5 cases in which operation was done, as well as in 4 other cases in which proved or presumptive pulmonary disease was present. Nonspecific inflammation was present in 4, but typical organisms were demonstrated in 3. These results suggest that bronchoscopy with bronchial biopsy could serve as a valuable diagnostic tool if properly utilized.

Weisel and Landis[15] examined 7 cases of pulmonary *Blastomyces dermatitidis* in which endobronchial lesions were seen during the acute, active phase of the disease. However, the symptoms most commonly seen were the bronchial lesion of superficial mucosal ulceration and the presence of a characteristic type of retained secretion in the involved bronchial segments. Bronchoscopy may be of help in the diagnosis of these lesions by the visual findings and also in providing material for bacteriologic identification of the infecting organisms. Pulmonary resection may prove gratifying in eradication of the localized pulmonary lesion due to *B. dermatitidis;* healing of the bronchial stump was satisfactory in the 2 cases reported. The pulmonary lesion of blastomycosis is usually one manifestation of a systemic disease. The patients described by the authors have responded favorably, at least temporarily, to iodide therapy, including administration by the aerosol route.

Foreign Bodies in the Tracheobronchial Tree

Miller[16] states that stereofluoroscopic guidance has made possible safe, easy removal of radiopaque foreign bodies from the bronchi when visualization or contact was impossible with ordinary bronchoscopy.

Monod et al.[17] believe that a skilled bronchoscopist with suitable equipment can remove as many as 98 per cent of aspirated foreign bodies, shortly after the accident; however, when such skill is not available, it may be difficult to extract a foreign body at a later date. Bronchial inflammation and edema, hypersecretion and infection, as well as bronchospasm, may make viewing and extraction of the foreign body exceedingly difficult. This is particularly serious when the patient is a young child, or in cases in which the foreign body is not radiopaque. Often, too, relative asphyxia may be present. Since unskilled attempts at bronchoscopic removal of a foreign body may result in serious laryngeal and tracheal edema, especially in infants, the authors propose that tracheotomy be considered particularly with large foreign bodies, with serious respiratory embarrassment and with gross bronchial hypersecretion, and when a number of attempts at bronchoscopic removal have failed. They believe that by this means the inflammation and edema may be lessened with improvement of respiratory embarrassment so that a further attempt is more likely to succeed.

DeNicola[18] reports the case of an unsuspected foreign body (surgeon's glove) in the lung removed after twelve years at the time of proposed lobectomy. Bronchoscopy in this case revealed only yellow, fluid pus, which continually obstructed the bronchoscopic view, and over 800 cc. of this material was evacuated from the right lung. No tumors or foreign bodies were seen. A reconstruction of past events indicated that a large surgeon's glove with the finger tips amputated had been used as a means of empyemic drainage in the first operation. This glove was filled with gauze packing that was gradually removed by a hos-

pital attendant — not the patient's surgeon. In the removal of the final strip of the gauze pack, the rubber-glove encasing was inadvertently allowed to drop into the chest cavity. The surgical removal of the foreign body resulted in complete and rapid healing.

Chalnot, Grimaud and Gille[19] have described surgical problems imposed by certain bronchial foreign bodies. In spite of the difficulties occasionally encountered in attempts to remove foreign bodies from the bronchi with the aid of the bronchoscope, specialists in extraction usually succeed in the end. However, in a few cases, surgery will be required. Two such cases are described in detail. In 1, symptoms of asphyxia followed unsuccessful attempts at bronchoscopic removal. In the other a chronic pulmonary suppuration developed about a foreign body that had remained unrecognized for two years and could not be removed through the bronchoscope. When the bronchoscopist meets with apparently insurmountable difficulties, endothoracic surgery may solve the problem.

Benign Tumors of the Bronchus

Soutter[20] gives a thirty-one-year hospital experience with the bronchoscopic approach to bronchial adenoma. He studied the use of the bronchoscope as a diagnostic and therapeutic instrument among 53 patients with carcinoid adenomas and 3 with cylindromas. Although the cylindromas are nearly always observable on bronchoscopy, the carcinoid tumors are occasionally too peripheral or not sufficiently intraluminal to be seen. As a rule bronchoscopic biopsy is an invaluable diagnostic measure. A pathological diagnosis in carcinoid adenoma is helpful when plans for the appropriate surgical resection are being made because these tumors can be cured by more conservative operations than would be effective in bronchogenic carcinoma. Although favorable results have been reported by others in the treatment of the carcinoid adenoma by transbronchoscopic resection, the results in 20 cases so treated in this series point to the opposite conclusion. Among patients who are good surgical risks for transthoracic resections, endoscopic removal should not be attempted for a number of reasons. In the first place the pulmonary infection beyond these tumors is usually a result of permanent pathologic changes that can only be cured by excision. Secondly, most of the adenomas are technically inaccessible for complete endoscopic removal. Thirdly, no malignant tumor, even of a low grade of malignancy, should be subjected to any surgical procedure less than total excision under direct vision. Fourthly, the operative mortality of bronchoscopic resection and that from infection in lung tissue beyond the adenoma are by no means negligible. Finally, the number of bronchoscopies and the number of years spent in treating patients by this means (an average of 15 procedures over three and a half years for the 20 patients) is unwarranted on the basis of prolonging suffering, the cost and the danger of metastases. The use of bronchoscopic aspiration and resection to relieve the symptoms of patients who cannot be expected to survive transthoracic resecting or to relieve them before thoracotomy is justified, effective and often compatible with prolonged survival.

Soutter, Sniffen and Robbins,[21] in reviewing their small experience with the cylindroma, have found that the histologic pattern of the tumor, characterized by ample evidence of mitoses and invasive qualities, has been reflected in its subsequent behavior. It is often so extensive as to be inoperable; it tends to recur, and may have distant as well as local metastases. The carcinoid adenoma has always been a therapeutic problem because histologically it displays unmistakable evidence of a malignant potential, but behaves clinically in a fairly innocuous fashion. In thoracic surgery the preservation of normal lung tissue is a primary consideration. Resections that will produce excellent survival rates and still conserve the patient's pulmonary reserve are ideal. Experience with local resections is limited, but encouraging.

Barrett and Thomlinson[22] do not write the term "adenomata" because this word has a precise meaning in general pathology, and these bronchial tumors have some of the features characteristic of adenomata and some of carcinomata. Clinically, they behave as benign tumors; histologically, they appear to be malignant. The authors reviewed a group of 34 patients who suffered from "adenomata" of the air passages. All patients were followed. Whatever name the pathologist may apply to the group, the surgeon should regard the tumors as benign and treat them accordingly.

Good and Harrington[23] report a case of asymptomatic bronchial adenoma. Their experience indicates that adenoma of the bronchus can exist for a long time without causing significant symptoms. Apparently, such a tumor continues to grow, and may ultimately call attention to itself by causing hemoptysis or symptoms of bronchial obstruction. They advocate the surgical removal of all circumscribed masses found in the lung, whether or not symptoms are present, provided the mass does not give roentgenologic evidence of calcification and cannot be proved to be metastatic from some malignant process elsewhere in the body. In this way they hope to prevent the development of pulmonary suppuration, which may result in a much more serious disease for the patient, and to eradicate bronchogenic carcinomas masquerading in the roentgenologic appearance usually attributed to benign conditions.

Jenny[24] believes that bronchoscopy should be done in the diagnosis of bronchial adenoma. He states that malignant degeneration is observed in approximately 10 per cent of bronchial adenomas.

Ward, Bradshaw and Prince[25] report a case of bronchial adenoma in a seven-year-old boy treated by pneumonectomy. A review of the literature of 8 cases

of bronchial adenoma reported in patients under fourteen years of age is included. A plea is made to treat bronchial adenoma as a malignant lesion with pneumonectomy or lobectomy as the only treatment available at present to cure the tumor.

Moersch and Harrington[26] state that bronchoscopy has always been and is still one of the most valuable aids in the diagnosis of bronchial adenoma. This is due to the fact that the majority of adenomas originate in the large-stem bronchi and consequently can be readily seen on endoscopic examination and are readily accessible so that tissue from the tumor may be removed for microscopical study. The higher rate of positive visualization and positive identification by microscopical examination than is ordinarily obtained in cases of bronchogenic carcinoma can be explained on the basis that bronchogenic carcinoma arises from the bronchial mucosa, in contrast to adenoma, which arises in the mucous glands of the bronchus, and consequently many more of the carcinomas will be peripheral in location and, therefore, less likely to be visible through the bronchoscope and less amenable to biopsy than adenomas are.

Hilton and Tanner[27] present the case of a woman in whom the lower lobe of the right lung was removed in 1948 because of lipoid pneumonia. The stump of the bronchus was removed in 1952, because of intractable cough and recurrent hemoptysis. The stump was found to be the site of a bronchial adenoma.

McBurney, Kirklin and Woolner[28] have made a study of metastasizing bronchial adenomas. A review of the literature concerning bronchial adenomas, together with a study of cases involving such tumors at the Mayo Clinic, has yielded 87 cases of bronchial adenoma in which metastasis occurred. Nine of these cases were encountered at the Clinic. This study demonstrated that metastasis takes place in approximately 10 per cent of all bronchial adenomas. Except under special circumstances patients who have bronchial adenoma are best treated by appropriate pulmonary resection.

Thomas[29] reports 57 cases of benign lung tumors. Forty-one patients had bronchial adenomas. Roentgenologic evidence of obstruction to lung segments, cavitation beyond the tumor or extrabronchial masses was an indication for bronchoscopic search. The treatment recommended is resection of the tumor and diseased lung. Endobronchial excision is hazardous and inadequate. There were 10 hamartomas. Of 4 vascular tumors, 1 was an angioma. Three patients had arteriovenous fistulas. Two female patients had endobronchial fibromas.

Young et al.[30] described 2 cases of bronchial hamartoma. In the first case bronchoscopy showed a bluish-red knobby tumor. The right middle lobe was resected. In the second case bronchoscopy disclosed a globular tumor arising from the posterolateral wall of the right intermediate bronchus. The right middle and lower lobes were removed.

According to Moersch, Donoghue and McDonald,[31] who studied 35 surgically proved cases of hamartoma of the lung, the tumor occurs twice as often in men as in women and is most frequently observed during middle age. Hamartoma was found under the pleura, in the interlobar fissure and deep in the substance of the lung itself. In 3 of the 35 patients the tumor was located within the lumen of the bronchus, and in 2 of these 3, the tumors were removed by endoscopic procedures; in the remaining 32 patients, the tumors were removed surgically. The hamartomas varied from 1.5 to 3.0 cm. in diameter; they were firm, solid, grayish white, lobulated and spherical or oval. Hamartoma produced few pulmonary symptoms. In 57 per cent of the patients under observation, the tumor was accompanied by a completely asymptomatic course of events. As a result of this study, it was concluded that if it were possible to establish a diagnosis of hamartoma of the lung in the absence of bronchial invasion it might be advisable not to remove the tumor surgically, especially since hamartoma seldom, if ever, undergoes malignant change. However, because of the difficulty of differentiating hamartoma from malignant tumors on the basis of clinical and roentgenologic observations, it is frequently necessary to remove the hamartoma surgically. Fortunately, tumors located in the lumen of the bronchus can be removed satisfactorily by endoscopic procedures.

Peabody and his associates[32] reported an intrathoracic hibernoma representing, it was thought, the nineteenth proved case of this interesting neoplasm, and only the third arising within the chest. No abnormalities were noted at bronchoscopy. (Hibernoma is the term applied to a rare tumor composed of multiloculated fat cells morphologically similar to, if not absolutely identical with, the cells of the so-called "hibernating gland" of animals.)

Ashmore[33] has reported a case of papilloma of the bronchus. At bronchoscopy a mass of pink tissue was seen in the right-lower-lobe bronchus; the right lower lobe was removed. The mucosa was heaped up in many frondlike projections. The pathological report was papilloma.

Schmidt, McDonald and Clagett[34] have seen 3 cases of amyloid tumor of the lower part of the respiratory tract and mediastinum. Two of the patients underwent pneumonectomy for this condition, since bronchogenic carcinoma could not be entirely excluded at the time of operation. The third patient was treated by bronchoscopic removal of part of the tumor. All 3 patients have done very well for more than five years since treatment.

Bronchogenic Carcinoma

Ochsner, Ray and Acree[35] have reviewed their experiences with 1457 cases of bronchogenic carcinoma. They state that cancer of the lung is increasing more than any other cancer in the body. They

believe that the unprecedented increase is due almost entirely to cigarette smoking. All men past the age of forty who have been heavy smokers should have a routine x-ray examination of the chest at least every six months. All shadows should be considered suspicious of carcinoma. Unfortunately, bronchoscopy makes a positive diagnosis in only about a third of all cases. By cytologic examination of the sputum, a correct diagnosis can be made in approximately 68 per cent of cases. In 20 per cent it is necessary to resort to thoracic exploration to make a diagnosis.

Essenberg[36] designed an automatic smoking machine to test the effect of cigarette smoke on the lungs of albino mice. The preponderance of tumors in the smoked mice exceeded those for the control mice by 31.9 per cent. He concluded that the preponderance of tumors in the affected mice was induced by the cigarette smoke.

Breslow et al.[37] state that the prevalence of lung cancer for men fifty to fifty-nine years of age is seven times greater among smokers than among nonsmokers and seventeen times greater among heavy smokers than among nonsmokers. Similar ratios were found for men aged sixty to sixty-nine.

Hammond and Horn[38] studied 187,766 men in an effort to determine the relation between human smoking habits and death rates. These authors believe that "all of the evidence we have seen seems to be consistent with the hypothesis that the association between smoking habits and death rates from lung cancer and diseases of the coronary arteries results from a cause and effect relationship." Deaths from cancer were definitely associated with regular cigarette smoking. Of the 844 deaths from cancer, 167 were indicated on the death certificates as being due to lung cancer. The death rate from lung cancer was much higher among men with a history of regular cigarette smoking than among those who never smoked regularly. Regular cigarette smokers had a higher death rate from cancer of sites besides the lung than men who never smoked.

An editorial comment[39] considers thirteen separate studies from six different countries, involving more than 5000 patients, all pointing to a correlation between smoking and cancer of the lung. Nonsmokers rarely have lung cancer, whereas the risk increases directly with the amount of tobacco smoked.

McGrath, Gall and Kessler[40] believe that multiple origins are more than sporadic occurrences in pulmonary neoplasms. To study this problem, large tissue sections were prepared from 87 lungs containing bronchogenic carcinoma. In 53 per cent of the cases, the major tumor contained 2 to 5 different histologic patterns arranged in such a manner as to imply initial independence and ultimate fusion of neighboring neoplasms. Such lesions cannot be recognized grossly and are important to the thoracic surgeon because they recur in the stump if he cuts across them.

Umiker and Storey[41] report the case of an adenocarcinoma developing in a peripheral bronchial adenoma. Proved adenocarcinomatous change in a bronchial adenoma further emphasizes the necessity of treatment by pulmonary resection.

Spohn[42] believes that up to 70 per cent of all tumors can be reached through the bronchoscope and that biopsies can be taken to determine whether a tumor is benign or malignant. Bronchoscopy has also been helpful in cases in which no shadows or outlines could be seen in the roentgenogram, and it has been especially valuable in cases in which irregularities were shown on the roentgenogram but no definite diagnosis could be established. The author especially mentions 2 patients, both of whom had similar symptoms; after bronchoscopy and biopsy it became apparent that 1 had an ulceration and stenosis of a bronchus caused by tuberculosis, whereas the second was shown to have a cancer of the lower-lobe bronchus.

McNulty[43] made a clinical follow-up study of 398 patients suspected of having lung cancer discovered in the Boston chest x-ray survey. The rate of case finding — namely, 39 primary cases of lung cancer out of 536,000 persons surveyed, or a little less than 1:14,000 — is depressingly low. Of even greater significance is the fact that of 22 patients whose lungs were resected only 5 were alive and well at the end of this follow-up period. Equally discouraging is the fact that resection could not be done in almost half the patients found so that the survey did not, in fact, as might have been hoped, uncover the disease in a significantly earlier phase than that seen in ordinary practice.

Umiker and Storey[44] have reported a case of bronchogenic carcinoma in situ, arising in an area of squamous metaplasia, with positive biopsy and cytologic examination of the bronchial aspirate treated by lobectomy. This article tends to emphasize the importance of squamous metaplasia.

Moersch and McDonald[45] have studied the significance of cell types in bronchogenic carcinoma. Their report is based on 1000 cases of proved bronchogenic carcinoma observed at the Mayo Clinic. The cell types of the tumors were as follows: small-cell carcinoma in 90 cases (9 per cent), adenocarcinoma in 137 cases (13.7 per cent), large-cell carcinoma in 378 cases (37.8 per cent) and squamous-cell carcinoma in 395 cases (39.5 per cent). The results of surgical treatment were more satisfactory in cases of squamous-cell carcinoma than in cases of the other types. The results of surgical treatment were least satisfactory in the cases of small-cell carcinoma.

Blades and McCorkle[46] have described a case of spontaneous regression of an untreated bronchogenic carcinoma. The diagnosis was made by exploratory thoracotomy and biopsy. The lesion was inoperable because of mediastinal spread. The slides were reviewed and checked, and no errors found. There was no explanation for the regression of this cancer, which represented the only known case of spontaneous regres-

sion of a bronchogenic carcinoma. Despite evidence of metastasis to the skull, the patient was in good health more than five years after the original diagnosis of bronchogenic carcinoma.

Wierman, McDonald and Clagett[47] have discussed a case of occult carcinoma of major bronchi. A cytologic examination of sputum disclosed malignant cells. Bronchoscopy revealed no tumor, but roughening of the mucosa on the medial wall of the right bronchus intermedius. Biopsy of this area showed a Grade 3 squamous-cell carcinoma in situ. Thoracotomy was performed on the right. The surgeon found the middle lobe and the anterior portion of the lower lobe to be quite indurated, and he removed the right lung. No gross tumor could be found in the surgical specimen. Numerous microscopical sections at various levels of the right main bronchus, bronchus intermedius, upper-middle-lobe and lower-lobe bronchi and to a varying degree their secondary branches revealed a diffuse, Grade 3 squamous-cell carcinoma in situ. Papanicolaou and Koprowska cite 4 cases in which surgical resection was performed. The tumors were not readily apparent in any of the 5 cases; 1 was in situ, and four were grossly minute lesions with a wide area of carcinoma in situ surrounding the invasive portion. Cytologic examination of the sputum disclosed malignant cells in all 4 cases in which this test was made. Malignant epithelium grows in a somewhat centrifugal manner. It obliterates the normal longitudinal furrows of the bronchial mucosa, imparting a granular or roughened appearance that can be detected bronchoscopically and that should be subjected to biopsy.

Pulmonary Adenomatosis

Storey, Knudtson and Lawrence[48] believe that so-called benign pulmonary adenomatosis and malignant terminal bronchiolar or alveolar-cell carcinoma are the same condition, varying only in the degree of malignancy in different patients or in separate portions of the same tumor. Among the various diagnostic methods employed in the recognition of these tumors, only cytologic study of the sputum and histologic examination of the excised lesion have proved to be of real value.

Hutchison and Fraser[49] state that pulmonary adenomatosis should be considered in any case in which there is abundant, relatively uninfected, clear, frothy sputum, and when the x-ray appearance suggests progressive consolidation in one lobe or multiple small opacities in one or more lobes. Cytologic examination of the sputum may be helpful and should certainly be done.

Farber et al.[50] have reported 4 new cases to the total of 217 described to date. In 2, bronchoscopy was performed, with negative results. Although they do not claim that cytologic study of the sputum will detect pulmonary adenomatosis with unfailing accuracy, the authors are convinced that attention to nuclear appearances will frequently suggest the possibility of adenomatosis, which can then be confirmed or disproved by other diagnostic studies.

Zheutlin, Lasser and Rigler[51] discuss a new diagnostic sign in bronchographic abnormalities in alveolar-cell carcinoma of the lung. They believe that the essential changes in the bronchographic pattern that should suggest the diagnosis of alveolar-cell carcinoma of the lung are as follows: a uniform, diffuse, marked narrowing of the segmental bronchi in the areas of lung involvement; rigidity and elongation of the bronchi; filling rather than coating of the bronchi; and a lack of filling of the terminal ramifications of the bronchi (appearance of the "leafless tree") or of the alveoli of the lung segments supplied by these bronchi.

Mears, Kirklin and Woolner[52] have reported 13 cases of alveolar-cell tumor of the lung in all of which bronchoscopy was performed. The gross findings at the time of bronchoscopy were significant in 8 cases. A frothy mucoid material was described coming from the involved segments of the bronchial tree in 5 cases, and some deformity of a bronchus was noted in 3. Biopsy was done at the time of bronchoscopy in 4 cases. In one of these positive evidence of carcinoma was found, and the pathologist suggested the possibility of an alveolar-cell tumor. Preoperative cytologic examination was done in 11 cases. Malignant cells were found in either the sputum or the bronchial washings or both in 7 (63.6 per cent) cases.

Cylindroma

Reid[53] has presented 5 cases of adenoid cystic carcinoma (cylindroma) of the bronchial tree with correlation of cases from the literature. This tumor has a long clinical course, occurs in males somewhat more often than in females and frequently involves the trachea. It is a tumor of moderately high malignancy, as evidenced by its pronounced infiltrative tendencies and the occurrence of distant metastases in a quarter of the reported cases. It is to be separated from both carcinoid tumors of the bronchus and from bronchogenic carcinoma.

Other Primary Malignant Bronchopulmonary Tumors

Grimes, Weirich and Stephens[54] report 2 cases of primary lymphosarcoma of the lung. In the first case bronchoscopic examination demonstrated that the tracheal carina was displaced to the left. An area of neoplastic tissue was observed in the proximal portion of the left-lower-lobe bronchus. Histologic study of a biopsy specimen obtained from this area showed only fragments of connective tissue infiltrated with lymphocytes. In the second case no bronchoscopy was done. The authors believe that primary lymphosarcoma of the lung can be treated effectively by extensive surgery alone or in combination with postoperative x-ray therapy.

Fojanini and Armenio[55] describe a case of fibro-

sarcoma in a twenty-six-year-old man. In this case a pneumonectomy was performed. The clinical picture resembles that of cancer, and only in cases in which a biopsy is possible can the diagnosis be established preoperatively.

Iverson[56] studied a case of bronchopulmonary sarcoma, which is most frequently a well circumscribed neoplasm composed of immature spindle cells; it occurs in middle life, is of relatively long duration and metastasizes in approximately a third of the cases.

Drewes and Willmann[57] describe their observations and treatment of 2 cases of primary sarcoma of the lung, and review the recent literature on the subject. The first case described was that of a man, forty years of age, who had polymorphic-celled sarcoma of the lung on the left side; the other was that of a nine-year-old boy with a spindle-cell sarcoma of the left inferior lobe. Successful surgery was performed on both.

Ehrenhaft and Womack[58] have reappraised mixed tumors of the lung, collecting 14 interesting cases. These tumors are most often composed morphologically of a mixture of tissues representing differentiation rather than modulation. Most commonly they occur in persons younger than those with squamous-cell cancer of the bronchus, and they are more frequently found in females. The symptoms and signs are usually those of mechanical obstruction of the bronchus, especially atelectasis with superimposed infection; occasionally, there is hemoptysis, and the symptoms are apt to be sudden in onset. There is early local invasion, but distant metastases are usually late so that extirpative surgery affords good results. These tumors are not considered adenomas; they should be regarded as malignant adenomas.

Tumors of the Trachea

Moersch, Clagett and Ellis[59] have studied 82 tracheal tumors, of which 47 could be classified as malignant and 35 as benign. Of the 47 primary malignant neoplasms there were 19 squamous-cell epitheliomas, 18 cylindromas, 3 hemangioendotheliomas, 1 myxofibrochondro-osteogenic sarcoma and 6 adenocarcinomas. Of the 35 benign neoplasms there were 22 papillomas, 8 cases of tracheopathia osteoplastica, 2 amyloid tumors, 2 xanthomas and 1 chondroma. The lesions may attain considerable size before giving rise to symptoms, or, if present, the symptoms may be of such indefinite character that they may be easily disregarded. Bronchoscopy is the most favorable aid in the diagnosis of tracheal tumors. It not only enables the examiner to remove tissue directly from the tumor for microscopical examination, but also makes possible the removal of secretions and smears from the tumor for cytologic study. In addition, it permits the exact localization of the tumor and, on occasion, its endoscopic removal.

Clark, Clagett and McDonald[60] studied 15 cylindromas of the trachea, which constitute the second most common malignant tumor of the trachea. The lesions have a distinct biologic course — that is, they grow slowly, they have a high rate of invasiveness and recurrence, and they have a definite potentiality for metastasis. Verified metastatic tumors occurred in 2 of these 15 cases and probably were present in 2 others. Two general methods of treatment were employed: surgical resection or excision and endoscopic diathermy or tracheotomy, with x-ray or radium therapy or both. Of the 15 patients 10 are dead. The majority of deaths were in patients whose tumors were treated with endoscopic surgical diathermy and x-rays. Among the 4 patients who are now in good health, the tracheal cylindromas were removed surgically by either tracheotomy and scissor excision or transthoracic surgical resection. The authors conclude that "a more positive approach to the treatment of this tumor should be taken in the form of early surgical excision when the tumor is surgically accessible."

Evans[61] has reported the case of a tracheal myxochondroma. This patient was treated by tracheotomy followed by resection of the involved tracheal wall. A full-thickness skin graft taken from the inner aspect of the right arm was reinforced with 28-gauge stainless-steel wire, after the manner of Gebauer, and sutured into the defect so that airtight closure was accomplished. Re-examination a year later showed normal vocal cords and a widely patent trachea.

Carr and Olsen[62] discuss several hypotheses regarding the pathogenesis of tracheopathia osteoplastica. They consider the most acceptable to be Dalgaard's concept, according to which undifferentiated connective-tissue cells in the wall of the trachea develop into cartilage cells that grow into islands of cartilage, which become partly calcified. According to the literature, tracheopathia osteoplastica usually does not produce any symptoms. Their experience is contrary to this, for all 7 of their patients had symptoms, which in some cases were serious; in fact, 3 of the 7 patients died, 2 because of complications of tracheopathia osteoplastica. Tracheopathia osteoplastica is a rare disease characterized pathologically by the development of growths of cartilage and bone in the walls of the trachea and bronchi and clinically by cough, expectoration, hemoptysis and bouts of obstructive pneumonitis. In 6 of the 7 cases the diagnosis was established by bronchoscopic examination. No specific therapy is available.

McKenzie and Rezek[63] reported a case of myosarcoma of the trachea associated with a Riedel struma. The patient, a fifty-four-year-old woman, was admitted to the hospital because of respiratory difficulty and a choking sensation of four weeks' duration. Bronchoscopy revealed a large mass obstructing the trachea approximately 20 cm. below the teeth. It seemed to be attached to the anterior wall of the trachea and extended down the left wall to the main-stem bronchus. A tracheotomy was done, and it was necessary

to introduce an extremely long tube to provide an airway past the tumor. A biopsy was taken, and the tissue was diagnosed as a malignant mesenchymoma. The patient was referred for x-ray therapy and a total dosage of 3000 r was given. Two weeks after completion of the radiation no evidence of the tumor could be found by bronchoscopic examination. The patient was discharged to outpatient care, and the tracheotomy tube was soon removed. Two weeks later respiratory difficulty recurred. In another two weeks she was readmitted to the hospital because of severe dyspnea. Bronchoscopy revealed a large tumor mass at the site of the original tumor. The tracheotomy was reopened, and the patient received additional deep x-ray therapy. The symptoms became worse, and sixteen days after the last admission she died. The autopsy diagnoses included, among other findings, primary sarcoma of the anterior wall of the trachea (myosarcoma) and Riedel struma; the thyroid gland was adherent to the trachea and surrounding structures.

Clagett, Moersch and Grindlay[64] believe that tracheal tumors are best diagnosed by bronchoscopy. The most common types are squamous-cell carcinoma, cylindroma, adenocarcinoma and hemangioendothelioma. Cylindromas of the trachea tend to grow slowly and to metastasize late, and thus are the most favorable type of tracheal tumor for surgical resection. Extensive experimental studies have resulted in the development of technics that permit surgical resection of some tracheal tumors.

Olson[65] reports a case of tracheal obstruction from benign lymphoid hyperplasia in a two-and-a-half-year-old girl whose first complaints were gasping for breath during exercise, a change in voice and noisy breathing while sleeping. A scout film of the upper airway revealed narrowing of the upper trachea as it merged with the larynx. The lumen of the trachea at this point was reduced a third the normal size by a mass arising from the posterior wall of the trachea. The child was hospitalized, and direct laryngoscopy was performed under light general anesthesia. A rounded, smooth, nonulcerated mass of firm consistence and with a broad base was seen in the posterior wall at the cricoid level. A 3.5-mm. bronchoscope could not pass the obstruction. Tracheotomy was performed. The tube was inserted at the level of the seventh and eighth tracheal cartilages. A 5-mm. bronchoscope was then introduced, and specimens of tissue were removed for microscopical examination. In a review of all the pathological material and the clinical history, it was believed that the patient had a benign deposit of lymphoid tissue beneath the tracheal mucosa.

Miscellaneous Conditions Affecting the Bronchi

Vinson[66] described 48 patients for whom the history, specialized examinations and subsequent observations seemed to justify a diagnosis of simple erosion of the wall of a small bronchus with pulmonary bleeding. Some patients had colds shortly before the bleeding began, and others engaged in fairly strenuous physical exertion within a day of the bleeding. There were 35 men and 13 women in this group, seventeen to sixty-five years of age. All were examined and treated bronchoscopically at the time of active bleeding, which usually occurred from the posterior or posterolateral division of the bronchus to the lower lobe. Treatment consisted of dilatation of the bronchus, mechanical curettement of the bronchial wall with an aspirating tube, instillation of a 1 per cent solution of phenylephrine hydrochloride or topical administration of thrombin and, finally, insufflation of a mixture of sulfanilamide and sulfathiazole powder into the affected bronchus.

Kergin[67] presented 8 cases of silicotic and tuberculosilicotic lesions simulating bronchogenic carcinoma. The patients were all men, and in each case clinical and radiographic evidence strongly suggested a diagnosis of bronchogenic carcinoma. Five patients were treated by pneumonectomy, and 1 by lobectomy, and in another thoracotomy was done and biopsies taken. The eighth patient was not operated on. A study of the excised tissue indicated that the fundamental feature of the pathology in this group of patients was complete or partial bronchial obstruction by pressure of lymph nodes, which were the site of a chronic inflammatory reaction secondary to inhalation of silica-containing dust.

Minton, Young and Shanbrom[68] have reported 2 new cases of endobronchial geotrichosis in which the chief complaint was cough with hemoptysis. Frequent x-ray studies of the chest revealed no parenchymal disease. Sputum examinations showed only pneumococci until a direct smear with potassium hydroxide demonstrated innumerable geotrichum spores. A culture grew the fungus luxuriantly. Bronchoscopic scrapings from the endobronchial lesions also revealed geotrichum. Iodide therapy appeared to benefit both patients.

Minetto and Daprà[69] believe that broncholithiasis probably occurs much more frequently than it is diagnosed. It is asymptomatic in many cases, and often a person who has never complained of pain suddenly coughs up a single broncholith. The broncholith may have a tuberculous origin. Although the hypothesis that broncholithiasis arises from a tuberculous lymph node is the one most generally accepted, any inflammatory process of the lung or bronchus may under certain conditions cause it. A broncholith may also form around organic or inorganic matter that arrived in the bronchial lumen through the respiratory pathways. This explains the presence of aluminum and silicon in some broncholiths. Another cause of broncholithiasis may be bronchial calcification.

Mears, Prickman and Moersch,[70] in a study of bronchostenosis and asthma, state that of 1034 consecutive asthmatic patients who underwent bronchoscopic examination, 327 had stenosis of one or more bronchi. Dilatation and aspiration alone usually gave

prompt relief. Bronchostenosis was far more frequent in the right than in the left lung. The superior division of the lower lobe was most often involved. Two patients died after bronchoscopy. In 13 of the 327 patients with bronchostenosis subcutaneous emphysema developed after bronchoscopy with dilatation. Immediate improvement after dilatation of the stenosed bronchus occurred in 60.5 per cent of the patients, delayed improvement in 63.6 per cent, and sustained improvement in 60.5 per cent. The corresponding figures in a control group of asthmatic patients without evidence of stenosis on bronchoscopy were 55 per cent, 48 per cent and 45 per cent respectively.

Chance, Devine and Schmidt[71] report a case of long-standing cicatricial stenosis of the trachea secondary to tracheotomy performed sixty-one years previously. The acute symptoms developed as a result of tracheitis or because of the fact that as the patient became older it was more difficult for her to satisfy her need for oxygen through the narrow slit. Relief of symptoms followed endoscopic dilatation. The complications occurring after tracheotomy are reviewed, and the various methods for the management of cicatricial laryngeal and tracheal stenosis are summarized.

Conley[72] has discussed the diagnosis and treatment of encrustations in the trachea and their relation to surgery of the head and neck. These encrustations are associated with the postoperative changes occurring in patients who have been subjected to tracheostomy. If small encrustations develop, aerosol trypsin or detergents should be incorporated in the program. If large encrustations occur, mechanical dislocation with the aid of a bronchoscope or by manual manipulation (in case of emergency) will have to be resorted to.

Shaw[73] has seen 10 patients with asthma or chronic obstructive bronchitis who had a distinct but undescribed clinical entity, mucoid impaction of the bronchi, which may closely simulate neoplasm, abscess or tuberculosis. It results when inspissated mucus accumulates in the second-order branch bronchi distal to a bifurcation. Shaw states that bronchoscopy is usually not helpful. The underlying asthma may lead to repeated episodes in other segments of the lung; resection should be limited to the actual lesion to preserve pulmonary function. At exploration it may be necessary to expose the bronchial wall to exclude neoplasm. The prognosis of this complication of asthma is the prognosis of the underlying disease. In 4 of the 10 patients pneumonitis has recurred in other segments and undoubtedly was due to mucoid impaction.

Trauma

Dalgleish, Hazen and Kinnear[74] report a case of traumatic rupture of a bronchus in a six-year-old boy that was complicated by severe subcutaneous emphysema. The resulting complete stenosis of the right main-stem bronchus was successfully treated by pneumonectomy two months after the acute episode.

Sauvage[75] describes a twenty-nine-year-old man in whom, after a crash injury to the chest, pneumothorax, subcutaneous emphysema and profuse hemoptysis with serious dyspnea developed. Continuance of respiratory embarrassment, pulmonary collapse and serous pleural effusion necessitated further hospitalization. Bronchoscopy was not performed until three months after the accident, when the right main bronchus was shown to be completely torn off just distal to its origin at the bifurcation of the trachea. Surgical repair was successfully done although it must have been difficult. At the last bronchoscopic observation, the continuity of the bronchial tree was normal, and the lung was re-expanded and functional. The interest of this case report lies in the emphasis on the importance of early bronchoscopy in this type of injury and the possibility of delayed repair of the bronchus.

Weisel and Jake[76] have reported a similar case in which a right bronchus was completely ruptured by external trauma to the chest in an automobile accident. In addition to other injuries the patient sustained multiple rib fractures and pneumohemothorax on the right. Bronchoscopy, performed four weeks after the injury, showed complete occlusion of the orifice of the right bronchus at the level of the carina, and it was believed that complete bronchial rupture had occurred, with sealing of the main-stem orifice. The occluded bronchus was aspirated and anastomosed to the trachea. Postoperatively, the lung expanded well; bronchoscopy showed good healing at the site of anastomosis. The most useful procedure in the diagnosis of bronchial rupture is bronchoscopic examination.

Gebauer[77] has presented a series of cases illustrating some of the aspects of the technical application of bronchial resection to a variety of tracheobronchial lesions. Sixteen cases were discussed and tabulated in which various lobes were salvaged by bronchial resection. The lesions included healed tuberculous stenosis with bronchial obstruction by a caseous mass.

(To be continued)

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MEDICAL INTELLIGENCE

A NEW TRIPLE-LUMEN TUBE FOR THE DIAGNOSIS AND TREATMENT OF UPPER GASTROINTESTINAL HEMORRHAGE*

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BALTIMORE

THE first successful attempt at controlling esophageal hemorrhage by balloon tamponage was reported by Westphal[1] in 1930. He treated 2 patients with a Gottstein sound distended with water, and applied the compression effectively for twenty-four to twenty-nine hours without any harmful consequence. The idea attracted little attention until 1947, when Rowntree and his associates[2] obtained good results in 2 patients using a Miller–Abbott tube with a latex bag attached to the distal end. Since then other papers have appeared in the literature attesting to the value of balloon tamponage in the treatment of variceal hemorrhage.[3-7] Most of the authors focused their attention on the two important problems of applying the pressure directly over the bleeding site and of keeping the balloon in the same position, once it had been accurately placed. Various modifications of the Miller–Abbott tube were tried, and fluoroscopy was frequently employed. Although successes were reported with a single balloon, completely satisfactory tamponage was not obtained until the ingenious double-balloon tube was described by Patton and Johnston.[8] They used a triple-lumen tube with a balloon that was inflated while in the stomach and pulled toward the diaphragm to localize the cardioesophageal junction. They thereupon inflated the second balloon, which was fixed to their tube just proximal to the first; in this way the varices were compressed by lateral pressure. The technic of using two balloons permitted both accurate placement and stabilization of the balloons during the period of tamponage. A similar tube later described by Sengstaken and Blakemore[9] has been used widely.

There are indications that blood enters the varix in patients with bleeding esophageal varices from two sources.[10] The major portion of the flow comes from the submucosal vessels of the stomach, which cross the cardioesophageal junction and anastomose with the esophageal submucosal veins. The second source of the blood is the periesophageal veins, which send perforating branches through the wall of the esophagus to communicate with the varices. These periesophageal veins also drain into the azygos, phrenic and intercostal vessels, which normally have low or even negative pressures, so that most of the blood flowing outside the esophagus probably drains toward the heart, rather than through the muscular coats of the esophagus into the submucosal varices. Furthermore, in some persons, the periesophageal veins are poorly developed, and flow through the submucosal vessels of the stomach must play an even greater part in supplying blood to the varix. Thus, although compression at the cardioesophageal junction may not mechanically occlude the bleeding varix, it prevents the delivery of blood from the submucosal gastric veins. Since the highest of portal pressures rarely exceeds 500 mm. of water[11] a great deal of pressure on the submucosal veins is not necessary. This probably explains why many of the single-balloon tubes have been successful in controlling esophageal hemorrhage.

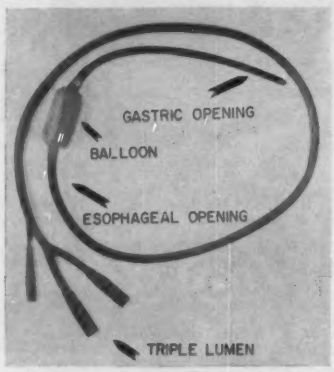

FIGURE 1. *Triple-Lumen Tube for Diagnosis and Treatment of Gastrointestinal Hemorrhage.*

Before any treatment can be instituted in the patient with a massive upper gastrointestinal hemorrhage, the diagnosis must be established. The many problems that arise have been the subject of numerous papers.[12-18] In recent years the sulfobromophthalein sodium retention test,[19] early x-ray studies[20,21] and esophagoscopy[22] have given valuable assistance in diagnosis, but each method has its limitations.

It appeared possible that the use of a tube that could functionally separate the contents of the stomach from that of the esophagus and would allow drainage of each compartment might be of value in localizing the bleeding site. A triple-lumen tube with a single balloon obstructing the cardioesophageal junction, and with two lumens for draining the stomach and esophagus, was devised for this purpose (Fig. 1).‡

For the past sixteen months the value of the tube has been explored in the Boston City Hospital. The first patient in whom the tube was passed had been

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receiving frequent paracenteses because of cirrhosis and was readmitted to the hospital with epigastric distress, hematemesis and melena. Blood was obtained only from the gastric lumen, in a patient who was obviously suspected of bleeding from esophageal varices. Subsequent x-ray studies confirmed that varices were not the source of the bleeding; a normal esophagus was demonstrated, and an ulceration of the lesser curvature of the stomach was found. In another striking case a patient in whom esophageal varices and a normal duodenum had been demonstrated roentgenologically eight months previously was admitted to the hospital vomiting bright-red blood and without abdominal pain. The triple-lumen tube indicated bleeding below the esophagus, and repeat x-ray studies revealed esophageal varices and a duodenal ulcer. In an alcoholic patient, in severe shock from a massive hemorrhage, attempts at diagnosis by either roentgenology or esophagoscopy were deemed unwise. After the tube had been passed, it became apparent that the bleeding site was infradiaphragmatic; in a fifteen-hour period, twenty-two transfusions were given, and a subtotal gastrectomy performed for a bleeding duodenal ulcer.

The tube described above has been used as an aid in localizing the source of the hemorrhage in relation to the diaphragm in patients with peptic ulcer, esophageal varices, gastritis and hiatus hernia. Its ease of application, safety, and assistance in diagnosis and treatment have made it useful in such cases.

At present, the tube has been passed in 20 patients, but was unsuccessful in 4 others who were either comatose or too unco-operative. Because excessive traction is not employed, once the tube is in place and the balloon inflated, there is no tendency for it to pull out. In these 20 patients, the impressions resulting from the use of the tube were later verified by x-ray, operative or post-mortem examination in 18 cases. In the remaining 2 the proof of the bleeding site that the tube indicated could not be definitely established.

Tamponage by the single balloon when esophageal hemorrhage has been diagnosed has not been uniformly successful. In 10 patients the hemorrhage was controlled, and in 3 others there was no beneficial effect. Balloon compression was maintained for two to sixty hours when control was effective. In the 3 failures the patients were bleeding so vigorously that even replacement of the single-balloon by the double-balloon tube could not control the exsanguinating hemorrhage. The relative effectiveness of these tubes in controlling esophageal hemorrhage remains to be determined.

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BY THE LONDON POST

Hospital Care — The Social Atom

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LONDON, ENGLAND

THE demand on the hospital service seems to grow each year. Nearly all hospitals can show a steady rise in their outpatient attendance figures, and, in spite of an increase of about 25,000 hospital beds since 1949, there is in many areas a long waiting time for nonurgent admissions.

The postwar development of new industrial and housing areas has also caused inadequate hospital facilities in many districts. For these reasons, the announcement by the Ministry of Health of a building program for new hospitals has been welcomed as being long overdue. It has also served to emphasize once more the vast cost of the National Health Service. In spite of a total annual expenditure on the service of more than £400,000,000 the amount spent on hospital building since 1951 has only been at a third of the rate prevailing before the war. Under nationalization the hospitals have certainly enjoyed greater resources for their running costs, but for capital investment they have fared far worse. Not one new general hospital has been built since 1939. The newly announced grants will provide increasing allowances for hospital expansion, rising to a total of £18,000,000 by 1957. Allowing for price changes this still represents little more than half the prewar rate.

Although admitting that the sums provided are not large, the Minister has argued that much of the expenditure on hospitals in the prewar years was on building small hospitals scattered over the country according to the wishes of the benefactors, local enterprise and local wealth. Now that all hospital expenditure is met from central funds there should be a better chance that the new building will take place where it is most needed and the money put to better use.

In addition to the grants for new hospitals the Minister announced the allocation of funds for the modernization of obsolete equipment. Such an allocation is also regarded as overdue, for it is generally recognized that the replacement of such equipment as antiquated boilers and laundries, must lead to a long-term economy in maintenance expenditure.

Apart from the cost of building and maintaining hospitals, the cost of staffing them is another major problem. Because of the relatively unattractive salary scales that a nationalized industry can offer, many hospital authorities are finding themselves unable to compete with industry in the search for nonmedical staff.

These and other problems are making many of the administrators, physicians and surgeons working in the service conscious of a sense of frustration. No doubt the problems will be solved in due course, and it is to be hoped that the report of the Guilleband Committee will form a basis for reviewing the service as a whole. This committee, appointed in 1953 to inquire into the present and prospective cost of the service, has made a thorough survey and has lately finished hearing evidence from every kind of body likely to be helpful. The report is now awaited with interest.

* * *

A mother recently wrote to the *London Evening News* complaining that her daughter, aged twelve, was wasting her time at school being solemnly taught how to clean her teeth, wash herself and make a sandwich, a jelly and a piece of toast. Though this mother was shocked, it is apparently a fact that many children are sent to school with no training in the basic principles of normal behavior. It has been suggested that this is because the Welfare Departments, the Health Ministry and the School authorities have assumed more and more of the duties and responsibilities that properly belong to an individual, his family and his home.

Dr. H. V. Dicks, in the *Lancet,* has recently reemphasized the importance of the home and the family in the life of the child. He has pointed out that the human infant is endowed more than any other living creature with the biologic need for the intimate experience of loving, protective mothering. For this reason the family must remain what he describes as the "social atom" — the indivisible unit of social organization. Anything that stands between the child and its mother, whether it is domestic strife or an overindulgent foster parent in the form of the State, may cause signs of deprivation to appear in the child.

Yet the *Times,* in a lighthearted leading article, accepts all this psychiatric advice with a cynical grain of salt, arguing that children who are growing up without particularly disliking one or the other parent and are free from any consuming jealousy of brothers or sisters may well be handicapped. They have no hope of achieving the state of genius. What biographer would waste time on a subject in whose infancy the most patient research, opening all the cupboards and prying into every bag, fails to find a skeleton or let out a carefully hidden cat?

How unfortunate in later life are those who have nothing to say when all about them are recalling hard-luck tales of infancy! Surely, they argue, it is the "bossy" sisters and the "bullying" brothers who strengthen the fibers of ego to enable the grown man to cope with the burden of the modern day.

Perhaps the proper mixture lies somewhere between the two — just enough bullying, just enough bossy sister and plenty of parental love so that the average child may receive enough stimulus to develop a personality of his own and avoid being stereotyped.

There are also those who think that too much is demanded of the present-day mother. Because of the lack of domestic help she must be housekeeper, cook and parlormaid in addition to performing her strictly maternal duties of caring for the childen. In this respect a recent correspondent to the *Times* lamented the ever-increasing disappearance of "Nanny" from the nurseries of our land. He recalled her code of manners and morals and of good and bad behavior and how the children of bygone days were kept under her strict control in the nursery. Nevertheless, it appears that mothering is the inescapable duty of women, and statistical proof of its importance is revealed in a recent report of the Social Medicine Research Unit of the Medical Research Council, as described in the *Lancet.* Stillbirth rates and mortality in the first month and in the ensuing eleven months of life are shown to be greatly influenced by the mother's age, the number of children previously born, her social class and where she lives.

It is the social differences that are of greatest interest because they are at least as great today as in 1911, though the infant mortality rate is much lower in all classes. Thus, the mortality among miners' babies in the first month is 70 per cent greater and that among older babies four times greater than that among babies of higher professional workers.

It is difficult to explain the persistence of this difference in spite of the leveling of social conditions in recent years. The authors of the report wonder whether the causes of the decline in infant deaths may not be distinct from the basic causes of class difference in mortality. The *Times,* commenting on the report, asks whether the underlying cause may not be found in social differences in maternal competence in the care of children.

CASE RECORDS
OF THE
MASSACHUSETTS GENERAL HOSPITAL

Weekly Clinicopathological Exercises

FOUNDED BY RICHARD C. CABOT

BENJAMIN CASTLEMAN, M.D., *Editor*

VIRGINIA W. TOWNE, *Assistant Editor*

CASE 41171

PRESENTATION OF CASE

A sixty-five-year-old woman entered the hospital in a semicomatose state.

Information concerning the present illness was obtained from the record of an admission at another hospital. During the five weeks before admission to this hospital, the patient intermittently had loose bowel movements, three or four times daily, and periodic episodes of nausea, with vomiting. At the time of onset Kaopectate, paregoric and a low-residue diet were prescribed. After seven to ten days of this therapy, she had failed to respond, and hospitalization was recommended but refused. The diarrhea persisted, anorexia increased, and weight loss and weakness developed. Two weeks before admission, Sulfathalidine (1.0 gm. every six hours) was given, without beneficial effect. She was admitted to another hospital, where she was co-operative, moderately alert, but vague about the details of her history. She was moderately cachectic, with signs of recent weight loss. There was a questionable icteric tinge to the scleras; the tongue was slightly enlarged and beefy red, with longitudinal fissuring. A few scattered rales were audible at the right-lung base. The cardiac rate was 92, the rhythm was irregular, and a radial pulse deficit was noted. The abdomen was relaxed, but hyperactive peristalsis was noted in all areas. It was mentioned that deep palpation produced a sensation of nausea. Urinalysis showed a + test for albumin. Examination of the blood revealed a white-cell count of 15,200 and a hemoglobin of 14.9 gm. per 100 cc. A stool was slightly positive to the guaiac test and a stool culture yielded alpha-hemolytic *Streptococcus faecalis*. The cephalin flocculation test was negative in forty-eight hours. A blood sugar determination two days before admission to this hospital was 147 mg. per 100 cc.; the nonprotein nitrogen was 78.5 mg. three days and 122 mg. per 100 cc. one day before admission. Therapy at the time of the initial hospitalization included intravenous administration of fluids, consisting of 2000 cc. daily, given as two-thirds 5 per cent dextrose in physiologic saline solution and one-third 5 per cent dextrose in water with added potassium solution. Two transfusions (500 cc. of whole blood each) were given. Nevertheless, loose stools (two to three times daily) continued. The general condition deteriorated. Two days before admission, she received 1.2 mg. of digitoxin. She was quite dyspneic, and a Kussmaul type of respiration developed, necessitating nasal oxygen. She became incontinent of urine, and during the twenty-four hours before admission the output of urine was virtually nil. On the morning before admission to this hospital, cystoscopy revealed a normal bladder, and a right retrograde pyelogram showed no abnormality. A barium-enema examination was reported as negative.

The past history and a review of the systems were noncontributory. There was no history of melena or previous gastrointestinal disorder.

Physical examination disclosed a poorly nourished and dehydrated woman, who was semicomatose and responded only briefly to simple questions and commands. The pupils were small, but reacted to light and accommodation. The fundi were not visualized. The mucous membranes were dry and beefy red. The tongue was contracted and fissured. The breath smelled faintly of acetone. The neck was voluntarily held rigid, and the neck veins were slightly distended. Inconstant, coarse, moist rales were heard at both lung bases, but more prominently on the left side. The left cardiac border was 10 cm. from the midsternal line. The rhythm was grossly irregular at a rate of 96. The abdomen was soft. Peristalsis was active, and was heard just under the precordium. No abdominal masses were palpable. Rectal examination revealed thrombosed external hemorrhoids. The knee jerks were hypoactive and the ankle jerks absent, but the neurologic examination was otherwise negative.

The temperature was 99.4°F., the pulse 80, and the respirations 24. The blood pressure was 116 systolic, 50 diastolic.

Urinalysis showed a specific gravity of 1.009, a ++ test for albumin and 250 red cells, 25 white cells with occasional clumps and a few hyaline and granular casts per high-power field in the sediment. Examination of the blood revealed a hemoglobin of 12.2 gm. per 100 cc. and a white-cell count of 9600, with a normal differential. On smear the red cells and platelets appeared normal. The nonprotein nitrogen was 225 mg. per 100 cc., and the sodium 134 milliequiv., the potassium 3.7 milliequiv., the chloride 103 milliequiv., and the carbon dioxide 16 milliequiv. per liter. A smear and culture of the urine were negative. An electrocardiogram demonstrated frequent ectopic ventricular contractions at times, with a bigeminal rhythm at a rate of 85. This arrhythmia,

along with sagging ST segments and delayed atrioventricular conduction, was thought to be consistent with digitalis effect and probable toxicity. On a single abdominal film no abnormal masses were seen; barium was distributed throughout the large bowel. An x-ray film of the chest showed some streaking at both bases, consistent with areas of atelectasis. A number of calcified scars were seen in both lungs. The heart was prominent in the region of the left ventricle, and the aorta was sclerotic.

Initially, therapy consisted of penicillin, streptomycin, paregoric and fluids via a Penrose tube. The patient excreted a small amount of dilute urine during the first hospital day and became more drowsy. On the following day the respirations were deep and increased. The mental status was essentially unchanged. Coarse, sticky rales were still present at both bases. The heart sounds were of fair quality, and the rhythm was still somewhat irregular. Pitting edema was now found in all dependent parts. The output of urine was slightly increased. The serum nonprotein nitrogen was 170 mg. per 100 cc., and the serum sodium 135 milliequiv., the potassium 2.9 milliequiv., the chloride 110 milliequiv., and the carbon dioxide 23 milliequiv. per liter. On the third hospital day, the patient was given intravenously 80 cc. of sodium lactate (one-sixth molar solution) and 20 milliequiv. per liter of potassium chloride in 1000 cc. of 5 per cent dextrose in physiologic saline solution. This was followed by a slow intravenous drip of 10 per cent dextrose in water. The white-cell count was now 19,300. A urinalysis showed a specific gravity of 1.011, a +++ test for albumin and rare red cells and occasional white cells per high-power field in the sediment. The blood nonprotein nitrogen was 203 mg., the total protein 5.4 gm., the albumin 3.1 gm., and the globulin 2.3 gm. per 100 cc., and the carbon dioxide 12 milliequiv. per liter. A guaiac test on a light-brown stool was negative, as was a second urine culture; a stool culture was negative for enteric pathogens. The temperature was 101°F., the pulse 92, and the respirations 32. The patient became deeply comatose during the day. Tube feedings were vomited, and the output of urine decreased to less than 100 cc. during the twenty-four-hour period. Peripheral edema was present, the lungs were unchanged, and the cardiac rate was still irregular, mostly without bigeminal rhythm. Respirations became increasingly shallow and rapid. She died on the fourth hospital day.

Differential Diagnosis

Dr. Marian W. Ropes*: There is very little that is definite about the patient except that she died of renal failure. The course was extremely rapid. Beyond that my attempts will be to determine what other systems were involved and to what extent they may or may not have been related to the renal disease, and to find out what the renal disease was. The cardiac involvement seems to have been mild; there was some irregularity, — extrasystoles, — which I think was evidence of digitalis toxicity, and I assume that she had mild coronary-artery disease. During the course of the final illness slight evidence of cardiac failure developed. I think that was unrelated to the remainder of the picture.

The lungs were interesting only from two points: the areas of apparent atelectasis, which in my mind were also unrelated to the main picture, and some so-called scars. I should be interested in seeing the x-ray films at this point, although I doubt if they will help me very much.

Dr. John F. Gibbons: These are films made at the other hospital, apparently when the patient was quite ill. The bladder is greatly distended on the first set of films, but on the next day, after the right kidney has been catheterized, the shadow in the pelvis is no longer seen. Two sets of films from the examination of the colon a day apart show what may be an area of narrowing in the sigmoid. I cannot be positive about that since I have no information about the fluoroscopist's report. I am not sure how distensible that area was, but I do not see that area of the colon distended in the film taken after the bladder was emptied; the sigmoid has collapsed, however, and there again seems to be some narrowing. I do not know whether the physicians tried to catheterize the left kidney during the pyelographic examination.

Dr. Daniel S. Ellis: They did not; they catheterized only the right kidney.

Dr. Gibbons: The renal collecting system is somewhat overdistended, but the cortex looks normal. On the barium-enema examination I cannot see the renal outline on either side because of the gas, barium and so forth. The films made here a day later are not of much help. The patient apparently was semicomatose most of the time, and films of the abdomen are blurred by respiratory motion. The colon contains a great deal of fluid mixed with barium; I do not see anything except that possibly narrowed area. I do not see any ulcerations or other abnormality along the wall. The films of the chest show what was described as atelectasis. I think that the film was made with the patient in the expiratory phase; that would account for the crowding of the vascular shadows. The left ventricle may be slightly enlarged.

Dr. Ellis: This patient was ill with diarrhea for six weeks before she consented to go to the hospital.

Dr. Ropes: I think I can say that the lungs showed relatively little that was related to the present illness.

The gastrointestinal tract is a much more difficult problem. The initial symptoms were those of gastrointestinal disease. The major problem is to decide whether or not the initial symptoms represented a sign of underlying disease, which progressed and involved the kidneys, or whether they represented the first and unusual manifestation of uremia. Either situation is

*Associate physician, Massachusetts General Hospital.

possible, and I am afraid I shall not be able to decide which was primary. One of the possible conditions that might relate the gastrointestinal disease and the renal disease is infection, but I can think of no generalized infection that would adequately explain this picture. I cannot believe that tuberculosis would manifest itself in both systems. I doubt if the patient had a generalized infection explaining the involvement of the gastrointestinal tract. In fact it seems as likely to me that the gastrointestinal symptoms were the early symptoms of uremia as that they represented a primary gastrointestinal-tract disease. In considering neoplasms I wondered about lymphoma, myeloma and hypernephroma and discarded them all.

Another condition that might cause this combination of symptoms is an underlying renal disease such as mild nephrosclerosis, which had not been very severe but had been greatly aggravated by damage to the kidneys by sulfonamides. The gastrointestinal symptoms occurred first, with the first definite evidence of renal failure after the administration of sulfonamides. It is unlikely that the majority of this picture was due to sulfonamide toxicity, but I shall not be surprised if there was evidence of lower-nephron damage. I suspect that there was underlying renal disease, such as moderately severe nephrosclerosis. I see absolutely no way to rule out either of those conditions.

If one considers all the conditions that rapidly produce renal failure, and also a severe constitutional reaction with marked weight loss, weakness and gastrointestinal symptoms, with a rapid course and death in five weeks, one would have to think of polyarteritis nodosa, although, as always, I hate to mention it in such a peculiar picture. It could explain the renal failure, but ordinarily the renal failure of polyarteritis nodosa is not so rapid, but is prolonged, usually with hypertension and striking involvement of other systems. It is conceivable that the gastrointestinal symptoms were the result of the vascular involvement of polyarteritis, but it seems unlikely.

One would also have to consider glomerulonephritis as an explanation for the whole picture. A diffuse glomerulonephritis, starting with evidence of uremia but without more findings in the urine and unassociated with a blood-pressure elevation, would be very unusual.

The possibility that a toxin other than a sulfonamide produced the gastrointestinal and renal symptoms has to be considered. Obviously, the picture is not that of mercury poisoning although it is not too different. It would be unusual for the gastrointestinal symptoms to be so severe so long before the renal disease was manifested. If this was mercury poisoning, by the time the patient died with renal disease she should have had more severe gastrointestinal findings, with necrosis and obvious oral and rectal lesions along with lesions in the remainder of the tract. On the other hand, the possibility of a poison in the wider sense of the word has to be kept in mind, although I can find none that explains the whole picture to my satisfaction.

One surely has to think of pyelonephritis, although there was little evidence for it. The cultures of the urine were negative; the urinary sediments were normal except for one after cystoscopy and retrograde pyelogram. Necrotizing papillitis, which has been thought to be a form of pyelonephritis, should be mentioned. There is a suggestion that the patient had diabetes, but very little information was given: no test of the blood sugar was done; no sugar in the urine was mentioned, which I assume means that the test was not positive. The blood sugar in the other hospital was 147 mg. per 100 cc., but she may have been receiving glucose at the time the blood was withdrawn for the test. She had an odor of acetone at a time when I think she was in renal failure, and she surely had other causes for ketosis. She had a very low carbon dioxide when the remainder of the electrolytes were apparently not very abnormal. The low potassium, which would be extremely unusual in renal failure of this degree, can surely be partly explained by the diarrhea and possibly by the urinary excretion of it. Diabetes has to be considered as well as necrotizing papillitis. In the presence of so little evidence for it in the urine — the negative cultures and very few cells — I should think it was extremely unlikely, as are the other renal lesions of diabetes. Kimmelstiel–Wilson disease rarely if ever results in such an acute course, with no preceding signs or symptoms and no evidence of chronic renal involvement.

Among the more unusual conditions, I am sure one has to consider amyloidosis in any patient who had gastrointestinal and renal disease. Ordinarily, however, the two manifestations of the disease are not found together. The rare case with gastrointestinal involvement and bleeding, in which there is amyloid in all the vessels of the intestines, is usually not associated with the type of amyloid that deposits so commonly in the kidneys and causes renal disease. The amyloidosis that may be associated with ulcerative colitis or ileitis is not common but has to be considered. So far as I know, however, there has never been a case in which the signs of the gastrointestinal disease did not appear until the time of renal failure from the amyloidosis. If the patient under discussion had amyloidosis, I think I should have to assume that it was primary, not secondary to other disease, and that the first signs were those of uremia, with diarrhea, nausea and vomiting going on to progressive renal failure. One of the points in favor of this is the urine. Amyloid is a type of renal disease in which one finds mostly albumin and relatively few cells in the urine.

Perhaps I should say something about other gastrointestinal-tract lesions. I cannot see how a neoplasm of the gastrointestinal tract could explain the combination of findings. I have mentioned the ulcera-

tive-colitis group. There is no question that a nonspecific enterocolitis unrelated to the final renal disease could have been present in this patient. I cannot, except for the slight connections that I have made, think of a disease that would relate these disorders more closely.

When I try to put this all together, I find it extremely difficult to decide between the two major diagnoses: whether the gastrointestinal symptoms represented a part of a renal disease that ended in renal failure or whether there was more generalized disease that included the gastrointestinal tract and renal system. I think it is conceivable that the whole picture could be seen with uremia alone; however, the diarrhea occurring two weeks before evidence of severe renal failure and persisting to this degree is surely uncommon. I do not know which to choose among the relatively common conditions such as nephrosclerosis, made worse by a toxin such as a sulfonamide; a diffuse disease such as polyarteritis nodosa, rare and with abnormal manifestations; a combination of enterocolitis and amyloidosis; and diabetes with a peculiarly rapid progression of the renal manifestations. Since I have to choose from this very motley array, I shall say that the gastrointestinal disease was related to the uremia, and I think that there is little to help me decide definitely what the cause of the uremia was. I shall choose the rare condition, amyloidosis, instead of one of the more common renal diseases.

Dr. William H. Baker: I should like to ask Dr. Ellis if there was any other information about this patient's renal failure during the first part of the illness.

Dr. Ellis: No. She was a native of New Hampshire and did not like hospitals. My impression is that her physician told me that she had had diarrhea for about three months before admission to the other hospital, and that she did nothing about it. In the month before admission to the other hospital, she lost weight and became weaker and weaker. I do not know whether a urinalysis was done before admission.

Dr. Alfred Kranes: Was the nonprotein nitrogen elevated when she was first admitted to the other hospital?

Dr. Ellis: Yes; she was critically ill the night she was admitted to the hospital in New Hampshire.

Dr. Benjamin Castleman: The first nonprotein nitrogen in the other hospital was 78.5 mg. per 100 cc.

Dr. Kranes: I am interested by the very low potassium. In view of the extraordinary degree of nitrogen retention, I wonder whether the gastrointestinal disease, whatever its nature, may not have been responsible for the renal failure on the basis of hypokalemic nephrosis.

Dr. Ellis: I was influenced by the telephone conversation I had with the physician and believed that the primary disease must have been of gastrointestinal origin. When the patient entered this hospital she was too sick to permit even a sigmoidoscopy. Obviously, by the time we saw her, as Dr. Ropes has pointed out, the immediate problem was one of renal failure. After having wondered whether she had pyelonephritis, we finally decided that she had, as the primary disease, a diarrhea, cause unknown, probably amebic or ulcerative colitis and nephrosclerosis.

Dr. Baker: Necrotizing papillitis is not always associated with diabetes. Is that not true?

Dr. Ropes: I did not mean to give that impression. Certainly, renal obstruction is the other condition with which it is commonly associated.

Dr. Charles H. Du Toit: With the one exception of the low potassium that Dr. Kranes has pointed out, the chemical findings were compatible with uremia alone. One would have expected considerably higher potassium values than were obtained. The first measurement was perfectly normal; the second was distinctly low. I suspect that the gastrointestinal tract as well as the kidneys contributed to the electrolyte disturbance.

Clinical Diagnoses

Uremia.
Nephrosclerosis.
Colitis, ?amebic.
Generalized arteriosclerosis.

Dr. Marian W. Ropes's Diagnoses

Uremia.
Disease of gastrointestinal tract as result of uremia.
Amyloid disease of kidney.

Anatomical Diagnoses

Vacuolar nephropathy, hypokalemic.
(Uremia.)
Chronic glomerulonephritis.
Lower-nephron nephrosis.
Ulcers of ascending colon, healing.
Acute colitis, left, uremic.
Anasarca.
Pulmonary edema.
Bronchopneumonia.

Pathological Discussion

Dr. Castleman: I do not know that I am going to answer this problem definitely either, Dr. Ropes. At autopsy we first found that the patient had a great deal of fluid — about 2 liters in each side of the chest, a liter in the abdomen and some peripheral edema and terminally a great deal of pulmonary edema and early bronchopneumonia. We examined the bowel very carefully for the cause of the chronic diarrhea. Localized in the right side of the colon were several shallow punched out ulcers, the largest of which measured 2.5 cm. in diameter. There was only a little erosion of the mucosa, and on gross inspection we wondered whether this was an amebic infection.

The left side of the colon over an area of about 15 cm. in length, on the other hand, had a very hemorrhagic appearance; the lesions there looked more acute than the lesions on the right side. The remainder of the bowel seemed to be within normal limits except for scattered petechial hemorrhages. Microscopically, the ulcerations of the cecum seemed old and healing, with many plasma cells, and were deep in the mucosa. Whether the patient had a dysentery or not we cannot say. We could find no amebas. I do not know whether it was an infection in the bowel that was healing by this time. The acute hemorrhagic process in the descending colon looked very active and was bleeding, and would fit with a uremic picture.

The kidneys, on gross examination, were somewhat reduced in size, each weighing about 125 gm., and presented a granular surface that we thought might represent either nephrosclerosis or chronic glomerulonephritis — an old process. Microscopically, the most striking change in the kidneys was in the convoluted tubules; the epithelial cells lining the tubules were tremendously vacuolated, filling the entire lumen of each tubule (Fig. 1). This vacuolar nephropathy was described some years ago by Dr. Kulka, of our laboratory, with Drs. Pearson and Robbins of the Boston City Hospital,[1] in a series of 15 cases of intestinal disease, such as chronic ulcerative colitis and regional enteritis. They did not know the cause of the nephropathy or how the gastrointestinal disease produced it. More recently, we have found that this peculiar vacuolar nephropathy may be associated with a low serum potassium. We had a similar case discussed at these conferences last year.[2] I believe, therefore, that this particular aspect of the renal disease was associated with the low potassium that the patient must have had over a long period. The patients with a low potassium and this nephropathy described in the literature usually have not died in uremia. The nephropathy has been an incidental finding, although it may have contributed to their deaths. Only in a few cases has it produced a nonprotein nitrogen as high as 150 mg. per 100 cc. Schwartz and Relman,[3] of Boston University School of Medicine, have studied this problem extensively and have recently performed renal biopsies on 2 patients with hypokalemia before and after treatment. The vacuolar nephropathy present in the original biopsy was not present in the biopsy after treatment.[4] Another lesion in the kidney was a mild but definite thickening of the glomerular capillary walls — a chronic intracapillary glomerulonephritis. Further adding to the renal insufficiency was a mild lower-nephron nephrosis characterized by granular and hemosiderin casts in the distal tubules, some peritubular reaction and evidence of regeneration of tubular epithelium. To reconstruct, I think that this patient had an underlying chronic glomerulonephritis, that either the sulfonamides or the transfusions that she received may have had something to do with the production of a mild lower-nephron nephrosis and that the final blow, the one that perhaps put her into uremia, was the low potassium, which produced the marked vacuolar nephropathy.

DR. DU TOIT: There was no evidence that she was a cascara addict, was there? That produces quite striking electrolyte changes.

FIGURE 1.

DR. JACOB LERMAN: Do such patients not have a typical picture in the bowel?

DR. CASTLEMAN: Yes; there is a melanosis. This patient did not have melanosis.

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CASE 41172

PRESENTATION OF CASE

A thirty-seven-year-old multipara was admitted to the hospital because of a "tumor" of the back.

For at least four years she had complained of episodic attacks of sharp, shooting pain involving the right side of the chest. Each such episode was thought

to originate in the right parasternal region but was associated with radiation straight through to the right side of the back. The bouts of pain were apparently fleeting and were unrelated to respiration. Because such attacks were repeated she had sought medical aid, but no abnormalities were demonstrated. For approximately a year she had been aware of a painless lump situated on the back just medial to the internal border of the right scapula. The lesion was not tender, and during the ensuing year slowly increased in size. She stated that it was always more prominent when the right arm was either extended forward or adducted across the anterior part of the chest.

The past history was noncontributory.

Physical examination was completely negative except for the mass in the back, located 6 cm. to the right of the midline posteriorly at about the level of the sixth rib. This was described as a smooth, nontender, diffuse bony swelling of the rib.

The temperature was 98°F., the pulse 82, and the respirations 20. The blood pressure was 130 systolic, 100 diastolic.

An uncatheterized specimen of urine had a specific gravity of 1.017 and gave a + + test for albumin; the sediment contained more than 100 red cells per high-power field. The hemoglobin was 13.8 gm. per 100 cc., and the white-cell count 7500. Review of a conventional posteroanterior film of the chest taken four years previously showed a suggestion of minimal rarefaction in the posterior portion of the right sixth rib (Fig. 1). However, the rib was not expanded, and the cortexes were intact at that time. An expansile radiolucent area was observed in the vertebral portion of the right seventh rib. Films taken on admission revealed marked expansion of the posterior portion of the sixth rib on the right for a distance of 9 cm. (Fig. 2). The expanded rib exhibited extensive new-bone formation and, in certain areas, a sun-ray pattern. There had been slight further expansion of the vertebral portion of the right seventh rib, which continued to show increased radiolucency rather than any new-bone formation. An operation was performed on the fourth hospital day.

Differential Diagnosis

Dr. John E. Gary*: I am confronted with a woman who harbored two tumors in adjacent ribs for at least four years and yet, for all practical purposes, was well. She had had no sustained pain and no constitutional symptoms, nor were there abnormalities in laboratory data that could be blamed on the tumors. Since the preoperative diagnosis centers on the x-ray films, I think we should look at them first.

In the first films of the chest, taken four years previously, there is thinning of the cortex of the posterior portion of the sixth rib, particularly along the lower margin, and small spicules of either calcium or bone in that segment of the rib (Fig. 1). The medullary canal is somewhat expanded. The changes are not striking, and they are even less so in the seventh rib. Four years later, in another film of the chest (actually the changes are better seen on the small films taken for rib detail), the seventh rib is changed little, if any. The sixth rib is now markedly swollen by a tumor, which is producing bone (Fig. 2). In the bone there are areas of radiolucency, the largest of which is at the anterior edge of the tumor, where the cortex is thinned and expanded but not destroyed. Farther back the cortex is missing, and the trabecular pattern is at right angles to the long axis of the rib. Closer to the vertebral end of the rib, where the tumor is tapering in size, the cortex is again seen. On this film no paravertebral mass is visible. The edge of the vertebra looks normal. The remainder of the skeleton is also normal.

I think that the lack of vertebral involvement and the fact that there was almost entirely bone production rather than bone destruction rules out infection — namely, tuberculosis and syphilis. If a blood Hinton test and tuberculin test were done it would be more certain.

Dr. Benjamin Castleman: I do not believe they were done.

Dr. Gary: Even without those tests I think infection is highly unlikely.

The first problem is to decide whether the tumor was primary in the bone or whether it was a metastasis. The only metastasis that even remotely looks like this lesion is from a renal-cell carcinoma. There was microscopical hematuria. Against such a diagnosis is the fact that the patient had no anemia in spite of the hematuria, and that only one of the two tumors changed in size in four years, the other changing hardly at all. Did she have an intravenous pyelogram?

Dr. Castleman: No.

Dr. Gary: Finally, metastases from renal tumors are very vascular. Was a bruit heard over the mass?

Dr. Richard H. Sweet: No.

Dr. Gary: I think that is an additional reason for excluding a renal metastasis. I also think it is a good reason for deciding that this was not a hemangioma. With hemangiomas, particularly so near the surface, there is a fair chance that a bruit would be audible. Also, a hemangioma produces a much coarser trabecular pattern than that seen in this case, which is quite fine. Eosinophilic granulomas occur in ribs, but so far as I know never grow this large; 3 or 4 cm. is about their maximum size. In addition, there was too much bone production for me to consider that diagnosis seriously. Sarcoidosis occurs in ribs, but again I think there was too much bone production, and I should expect the small cystlike areas of rarefaction to have been larger and rounder. In addition the fact that the hili were normal both four years previously and on admission is against sarcoidosis.

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Giant-cell tumor is not so easy to rule out. That is usually solitary, and here there were two lesions. Also, if this had been a giant-cell tumor, the areas of rarefaction would have been larger, the tumor would have been more cystic, and there would not have been so much internal bone structure as there was in this case. I should rule out a plasmoma and the malignant lymphomas on the same basis — namely, that there was so much bone production.

That brings me to the condition that I think has to be considered seriously in this case — that is, a cartilaginous tumor. Cartilaginous tumors are by far the commonest primary tumors seen in ribs. They are usually slowly growing; in fact, in over half the cases reported in the literature, a mass had been present for over two years before the tumor was excised. Cartilaginous tumors are frequently multiple and frequently involve adjacent ribs. When they are seen early, they are lytic, fitting very well with the appearance of this tumor four years previously, when it was almost entirely a lytic lesion. Later, varying amounts of bone production occur. Generally, the more bone production in these tumors, the less likely they are to be malignant — that is, in the ordinary sense of the word malignant. They do, however, have a tendency to recur unless they are widely removed with the capsule or periosteum still intact.

In summary I think that the patient had a chondroma, with a great deal of bone formation and perhaps areas of sarcomatous degeneration, particularly

FIGURE 1. *Posteroanterior Roentgenogram of Chest Taken Four Years before Admission, Showing Minimal Rarefaction of the Posterior Aspect of the Right Sixth Rib (Arrow).*

on the surface of the tumor. Even if there was no microscopical evidence of a malignant change, I think it should have been treated with respect because of the great tendency for local recurrence unless it is widely excised. I think she had a small chondroma in the seventh rib.

DR. GRANTLEY W. TAYLOR: I am impressed and pleased with Dr. Gary's review. He did not touch on osteogenic sarcoma, to which I should allude in passing. The leisurely progress of the tumor is against that diagnosis. I am inclined to agree with Dr. Gary. I do not think I should dismiss quite so rapidly the possibility of a metastatic renal-cell carcinoma. I think an intravenous pyelogram would have been highly desirable before operation.

DR. GARY: Yes; the septate form of metastases from renal-cell carcinoma can look a good deal like this. I think the facts that there seems to have been a lesion there four years previously and that there

FIGURE 2. *Posteroanterior Roentgenogram of the Right Ribs on Admission, Showing an Expansile Bone-Producing Tumor of the Right Sixth Rib, with Areas Exhibiting a Sun-Ray Pattern.*

were two lesions, one of which had not changed and one of which had grown, are good reasons for discarding that diagnosis.

DR. CASTLEMAN: Osteogenic sarcomas of the ribs are extremely rare. The tumors of the rib that years ago were called osteogenic sarcoma, we now know, were probably manifestations of fibrous dysplasia.

DR. GARY: Fibrous dysplasia is also something that should be mentioned, but again there was so much bone production that it is unlikely.

CLINICAL DIAGNOSIS

Osteochondroma of rib.

DR. JOHN E. GARY'S DIAGNOSES

Chondrosarcoma of sixth rib.
Small chondroma of seventh rib.

Anatomical Diagnosis

Hemangioma of rib.

Pathological Discussion

Dr. Castleman: Dr. Sweet, who operated on this patient, removed the fifth, sixth and seventh ribs (Fig. 3); only the sixth rib contained tumor. The tumor compressed but did not invade the rib below as well as the rib above it. The tumor, which measured 8.5 cm. in length and 3.5 cm. in maximum diameter, had expanded the rib to produce a very thin cortex. On section the tumor was trabeculated with soft, deep-red, almost purple areas separated by spicules and bands of bone (Fig. 4). It reminded me of a hemangioma in the pubic bone that I had seen a few years ago, and the microscopical examination of this case also proved to be a hemangioma. The calcified areas proved to be well formed bone. I suppose that the large amount of bone prevented the development of a bruit.

This tumor is a relatively rare bone lesion, occurring most often in the vertebras. This is the first one we have seen in the rib. We had the one in the ramus of the pubis and another in the fibula.

Dr. Sweet: There were two striking things about the patient, one being the severity of the pain and the other the localized tenderness. I think that many of us did not recognize the presence of the tumor in the films taken four years before admission. Dr. Joseph Hanelin, in our Department of Radiology, made the diagnosis of hemangioma before the operation, but I would not accept it. I thought it was probably an osteochondroma and treated it as such. I removed the entire sixth rib in which the tumor was located on the chance that there was a focus in the other portions of the bone. I took out the posterior portion of the seventh rib because of the x-ray appearance and

Figure 3.

because at operation that area looked peculiar, although I could not identify what it was.

Dr. Castleman: We found no hemangioma in the seventh rib; that showed nothing at all.

Dr. Sweet: It did not look like tumor when I excised it.

A Physician: I wonder what caused the x-ray appearance.

Dr. Castleman: Could the articular facet have been compressed in some way and given that appearance?

Dr. John F. Gibbons: All we can say is that the vertebral end of that rib looked more bulbous and less dense than the other ribs so we thought there was something other than normal bone in the rib. I should like to say that so far as I am concerned the radiographic appearance is much more like a chondromatous tumor than a hemangioma. We have one hemangioma of the rib in our teaching collection, which has an appearance identical with the lesion of the pelvis that Dr. Castleman described, and not one of the few hemangiomas that I have seen produced so much dense bone.

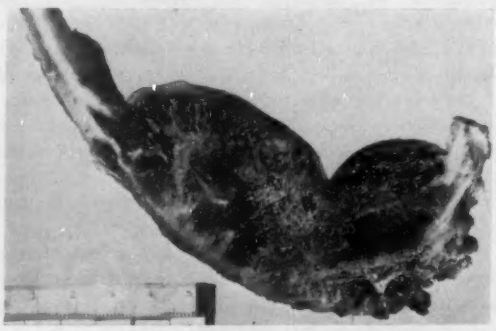

Figure 4.

Dr. Castleman: When a cartilaginous tumor degenerates, it calcifies but as a rule does not produce so much mature bone; here, there was a great deal of bone.

Dr. Sweet: Knowing that I was to discuss this case, I looked up the rib tumors that I had excised. There were 16 cases that I thought preoperatively were primary rib tumors; 2 were metastases — 1 from the thyroid gland and the other from an unidentified adenocarcinoma. Excluding those, the primary ones were 2 chondromas, 2 chondrosarcomas, 2 osteochondromas, 2 of fibrous dysplasia, 1 eosinophilic granuloma, 1 of chronic osteitis, 1 plasma-cell myeloma, 1 hemangiosarcoma, this hemangioma and a congenital malformation. There is a tremendous variety. Yet I believe, as I intimated a moment ago, that unless one is sure of the diagnosis on the basis of the x-ray appearance they should all be treated as though they were malignant. The mistake of cutting too close to a cartilaginous tumor is devastating to have to watch when the recurrences in the pleural cavity become manifest.

Dr. Gary: I should like to ask Dr. Castleman if he would not lump 6 of Dr. Sweet's tumors together — as being cartilaginous — the 2 chondromas, the 2 osteochondromas and the 2 chondrosarcomas.

Dr. Castleman: Yes.

The New England Journal of Medicine

Official Organ of
THE MASSACHUSETTS MEDICAL SOCIETY

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MICROFILM volumes available to regular subscribers through University Microfilms, 313 N. First Street, Ann Arbor, Michigan.

MATERIAL, other than original articles, should be received not later than noon on Thursday, three weeks before date of publication.

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COMMUNICATIONS should be addressed to the *New England Journal of Medicine*, 8 Fenway, Boston 15, Massachusetts. Telephone KE 6-2094.

MANUSCRIPTS, including references or bibliography, should be typewritten double spaced and submitted as original copy. References should conform to the style of the *Quarterly Cumulative Index Medicus* (listing name and initials of author, title of article, journal, volume number, first and last pages and year), and should be cited numerically in the order in which they appear in the text; the number should be limited to the absolute minimum.

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HUNDRED AND SEVENTY-FOURTH ANNUAL MEETING

THE Massachusetts Medical Society, on May 17, 18 and 19, will hold its hundred and seventy-fourth annual meeting — at the Hotel Statler in Boston, as has been the custom in recent years. Since the program is to be found elsewhere in this issue of the *Journal*, a few details only need be published in this column. As usual the supervising censors will meet at 4:30 p.m. on Monday, May 16, after which the annual meeting of the Council will be called to order at 5:00 o'clock instead of the usual later hour, and will temporarily adjourn for the Cotting Supper at 7:00.

The first general session of the scientific and clinical program will open at 9:00 a.m. on May 17 and the first luncheons of the specialty sections, now 12 in number, will be served that noon. At 2:30 the annual business meeting of the Society will take place, during which the presentation of awards to the outstanding fourth-year medical student of each of the three Massachusetts schools, and of badges to those fellows of the Society who have within the twelve months completed fifty years in practice will be made. The president will deliver his annual message on the state of the Society, and the hundred and forty-sixth discourse will be delivered by Dr. Carl Bearse, editor-in-chief of the *Norfolk Medical News*, on the vital subject "The Involuntary Plight of the Voluntary Hospital."

On Wednesday, May 18, after the morning panel discussion, the sixty-fifth Shattuck Lecture will be presented on "The Portal Circulation" by Dr. Charles G. Child, III, professor of surgery, Tufts College Medical School, and director of the First (Tufts) Surgical Service at the Boston City Hospital. The Shattuck Lectureship was established in 1890 as the result of a bequest that came to the Society on the death of Dr. George Cheyne Shattuck in 1854. That same evening at the annual dinner, the fellows of the Society and their guests will be addressed by His Excellency, Christian A. Herter, governor of the Commonwealth of Massachusetts. On the following day the scientific program will be concluded.

The Woman's Auxiliary to the Massachusetts Medical Society will meanwhile have been holding its own autonomous sessions at the Hotel Vendome. These will consist of the annual meeting at 10:00 a.m. on May 17, with Mrs. A. J. A. Campbell presiding, a luncheon on the same day with the Reverend Frederick M. Meek, D.D. as guest speaker, and an executive-board meeting and sherry party in the afternoon. On the following morning buses will leave the Hotel Statler for a conducted tour of the Harvard Yard. The Hi Spot will be hit that afternoon at the Hotel Continental, in Cambridge, with Hi Jinks, accompanying "Ladies In Waiting," an original musical extravaganza — book by Woodall and music by Costanza. The annual dinner of the Society in the evening will be shared on a coeducational basis.

Accompanying events of varying significance will be the annual golf tournament and the annual meeting of the Massachusetts Medico-Legal Society, coexisting in the afternoon of May 18, and the exhibition of works of art of the Massachusetts Physicians Art Society, an attraction that will last throughout the convention. Finally, and definitely educational in value, are the scientific and technical exhibits, presenting the results of patient research and displaying various new and astonishing remedies garnered from the most remote recesses of the known world.

WILLIAM BINGHAM, II

THE cause of medicine lost a good and important friend in the recent death of William Bingham, II. Born in Cleveland in 1879, he had made his home in Bethel, Maine, since 1911. In a sense, he had returned to New England, for the Bingham family first settled in Norwich, Connecticut, in the seventeenth century, soon removing to Andover, Massachusetts, where they continued to live for five generations.

Mr. Bingham's philanthropies were many and widespread, chief among them being the Bingham Associates Fund, the New England Center Hospital and the Gould Academy in Bethel, Maine.

The Bingham Associates Fund, whose broad purpose is the advancement of medicine, he named and founded in 1932. It was this fund that made possible the Bingham Program for regional medical care. The Fund became the keystone of Mr. Bingham's varied charitable activities in medicine in New England; through it, much of the New England Medical Center as it is now known came into being.

The Bingham Program is concerned with the problem of elevating the quality of medicine. Differences in medical opportunities exist between the modern medical center and the isolated practitioner. Essentially, the challenge that the Bingham Program accepted for itself has been the bridging of this existing gap on a regional basis. The New England Center Hospital in conjunction with Tufts College Medical School and the other units of the Center, serves as the base for the medical service and the postgraduate educational activities of the Bingham Program. In its relation with the physicians of New England, the hospital, as a reference unit, has joined some of the special features of group clinical practice with those of the university medical center.

In addition to his large support for the regional program and the New England Medical Center, Mr. Bingham's special interest in medicine led him to make substantial gifts not only to hospitals in Maine, such as the Rumford Community Hospital and the Central Maine General Hospital, but also to medical institutions elsewhere, such as the Columbia–Presbyterian Medical Center in New York and the Admiral Bristol Hospital in Istanbul, Turkey. These are but examples of his many charities in the field of medicine, which also included scholarships to needy students of medicine and aid to patients for medical care. Mr. Bingham did this unobtrusively, living quietly and giving in kindness and usually anonymously for the betterment of mankind through the advancement of medicine.

It may be that Minerva Medica has moved to America in recent years not only because she had so many loyal and talented devotees here in medicine but also because she had, outside the profession, so many warm and generous friends who could provide her with the means to create a truly suitable temple in which she could long thrive and through which her good deeds could spread to all the peoples of the world. Among the warm and generous friends of Minerva Medica, William Bingham, II, stood high indeed.

PRINCIPLES OF MATERNAL WELFARE

THE Section on Obstetrics and Gynecology of the Massachusetts Medical Society, inspired by its dynamic secretary, the late Dr. Raymond S. Titus, inaugurated on January 7, 1937, the publication of a weekly column in the Society section of the *Journal*. The column was devoted largely to brief obstetric case histories illustrating abnormalities of pregnancy and of labor. Many of them were candid discussions of errors in obstetric judgment or of faulty technics in delivery; all of them were of great educational value.

The publication of these cases under the aegis of the Section continued at weekly intervals, rain or shine, for five years, when, on January 22, 1942, the recently established Committee on Maternal Welfare of the Society, also with Dr. Titus as secretary and editor, took over the task. For a number of weeks a running analysis of causes of maternal deaths in Massachusetts during 1940 was published, followed by other subjects pertaining to obstetric crises. The publication of case histories was taken up again in March, 1942, continuing until August, 1943, when war pressures caused its abandonment.

In the present issue of the *Journal* the Committee, now under the chairmanship of Dr. John F. Jewett, has resumed its educational program after a lapse of nearly twelve years. The initial contribution consists of a set of minimum standards of obstetric care drawn up by the Committee. This will be succeeded by a series of illustrative cases, as in the old days, at approximately fortnightly intervals.

PHILIPPINE JOURNAL OF SURGERY, OBSTETRICS AND GYNECOLOGY

THE *Philippine Journal of Surgery*, edited by Dr. Luis F. Torres, Jr., has announced that beginning with the issue of January and February, 1955, it has come under the joint sponsorship of the Philippine College of Surgeons and the Philippine Obstetrical and Gynecological Society, and will serve as the official publication of the two organizations.

The new name of the periodical is the *Philippine Journal of Surgery, Obstetrics and Gynecology;* its

ownership remains with the Philippine College of Surgeons, and its volume numbering will continue as before. The first volume under the new name will thus be Volume 10 of the series.

The New England Journal of Medicine, having grown old in a relatively new civilization, congratulates the *S.G and O.* of the Pacific on its first few years in an old one. It wishes its expanded program success in its new Manila envelope!

The next annual meeting of the Superintendents of American Lunatic Asylums will be held in Boston on the fourth Tuesday of May current.
Boston M. & S. J., *May 3, 1855*

MASSACHUSETTS DEPARTMENT OF PUBLIC HEALTH

CONSULTATION CLINICS FOR CRIPPLED CHILDREN IN MASSACHUSETTS

Orthopedic Clinic	Date	Clinic Consultant
Salem	May 2	Paul W. Hugenberger, M.D.
Gardner	May 3	Carter R. Rowe, M.D.
Haverhill	May 4	William T. Green, M.D.
Greenfield	May 5	Charles L. Sturdevant, M.D.
Lowell	May 6	Albert H. Brewster, M.D.
Lynn*	May 9	Daniel M. Killoran, M.D., and John J. Crowley, M.D.
Brockton	May 12	George W. Van Gorder, M.D.
Springfield	May 17	James D. Fisher, M.D.
Pittsfield	May 18	Edward J. Coughlin, Jr., M.D.
Worcester	May 20	John W. O'Meara, M.D.
Fall River	May 23	David S. Grice, M.D.
Hyannis	May 26	Paul L. Norton, M.D.

Rheumatic-Fever Clinic	Dates
North Reading	May 3, 10, 17, 24, 31
Fitchburg	May 4, 11, 18, 25

Plastic Clinic	Held	Consultant
Eastern Massachusetts	Semi-monthly	Bradford Cannon, M.D.
Western Massachusetts	Monthly	Joseph M. Baker, M.D.

Physicians referring new patients to clinic should get in touch with the district health officer to make appointments. Patients are seen by appointment only.

*This clinic serves Lynn, Lynnfield, Saugus, Nahant, Revere, Chelsea, Everett and Winthrop. Referrals should be made directly to the Lynn Clinic.

COMMUNICABLE DISEASES IN MASSACHUSETTS FOR MARCH, 1955

RÉSUMÉ

Disease	Mar. 1955	Mar. 1954	Seven-Year Median
Chancroid	2	3	3
Chicken pox	2589	2045	2405
Diphtheria	3	4	10
Dog bite	1005	1184	864
Dysentery, bacillary	25	18	9
Encephalitis, infectious	5	8	5
German measles	577	293	351
Gonorrhea	189	156	174
Granuloma inguinale	1	1	0
Hepatitis, infectious	125	173	24*
Lymphogranuloma venereum	1	3	1
Malaria	1	2	0
Measles	11388	509	1835
Meningitis, meningococcal	4	10	7
Meningitis, Pfeiffer bacillus	3	4	4
Meningitis, pneumococcal	1	1	3
Meningitis, staphylococcal	0	0	0
Meningitis, streptococcal	0	0	1
Meningitis, undetermined	9	5	5
Mumps	1797	3024	1758
Poliomyelitis	1	7	1
Salmonellosis	22	40	11
Scarlet fever	1131	771	653
Syphilis	207	185	185
Trichinosis	0	0	0
Tuberculosis, pulmonary	140	201	203
Tuberculosis, other forms	9	11	15
Typhoid fever	0	2	1
Undulant fever	1	1	1
Whooping cough	200	338	241

*Six-year median.

Comment

Diseases below the seven-year median were diphtheria, meningococcal meningitis, typhoid fever and whooping cough.

Diphtheria now appears to be stabilized between 20 and 30 cases per year after dropping gradually over the past thirty years. This figure probably will continue unless the remaining number of susceptible persons is greatly reduced by immunization, particularly among the older age groups.

A similar situation exists with typhoid fever; a decline has been evident for a longer time with a present stabilized rate of 20 to 30 cases a year. However, in this disease where cases come from unrecognized typhoid carriers the future looks brighter because these carriers are dying rapidly and are not being replaced.

Diseases above the seven-year median were bacillary dysentery, German measles, infectious hepatitis, measles, salmonellosis and scarlet fever.

Bacillary dysentery and salmonellosis showed increases last month after being at lower levels the preceding two months of the year.

Measles, though still at a near-record level last month, showed a decline from the all-time high reached during the previous month.

Last month scarlet fever was at the highest point for any month since March, 1949.

Geographic Distribution of Certain Diseases

The following diseases were reported from the communities named:

Diphtheria: Boston, 2; Lynn, 1; total, 3.

Dysentery, bacillary: Boston, 20; Waltham, 3; Worcester, 1; Wrentham, 1; total, 25.

Encephalitis, infectious: Falmouth, 1; Fitchburg, 1; Malden, 1; New Bedford, 1; Worcester, 1; total, 5.

Hepatitis, infectious: Abington, 1; Arlington, 1; Attleboro, 1; Auburn, 4; Bedford, 1; Belmont, 1; Beverly, 2; Boston, 8; Brockton, 1; Brookline, 1; Cambridge, 3; Chelmsford, 2; Chelsea, 1; Chelsea Naval Hospital, 15; Concord, 1; Everett, 4; Fall River, 10; Fitchburg, 1; Fort Devens, 1; Foxboro, 1; Georgetown, 1; Holden, 1; Lexington, 1; Lowell, 3; Lunenburg, 1; Medford, 1; Newton, 2; Norfolk, 1; Northfield, 1; Norwood, 1; Palmer, 2; Peabody, 1; Plymouth, 1; Quincy, 4; Randolph, 1; Revere, 2; Seekonk, 1; Somerville, 1; Springfield, 8; Taunton, 1; Watertown, 3; Wellesley, 7; Wenham, 2; West Springfield, 5; Westover Air Force Base, 1; Weymouth, 1; Winchester, 1; Worcester, 5; Wrentham, 5; total, 125.

Lymphocytic choriomeningitis: Lawrence, 1.

Malaria: Westover Air Force Base, 1.

Meningitis, meningococcal: Ayer, 1; Beverly, 1; Boston, 1; Boylston, 1; total, 4.

Meningitis, Pfeiffer bacillus: Plymouth, 1; Quincy, 1; Shirley, 1; total, 3.

Meningitis, pneumococcal: Springfield, 1.

Meningitis, mumps: Natick, 1; Revere, 1; total, 2.

Meningitis, undetermined: Boston, 2; Chelsea Naval Hospital, 1; Framingham, 1; Leicester, 1; Pembroke, 1; Pittsfield, 1; Quincy, 1; Westover Air Force Base, 1; total, 9.

Poliomyelitis: Boston, 1.

Psittacosis: Pelham, 1.

Salmonellosis: Attleboro, 1; Beverly, 1; Boston, 3; Clinton, 1; Fall River, 1; Lawrence, 2; Marblehead, 1; Medford, 1; Newton, 2; North Reading, 2; Peabody, 1; Salem, 2; Sharon, 1; Springfield, 1; Wellesley, 1; Worcester, 1; total, 22.

Septic Sore Throat: Boston, 14; Cambridge, 5; Chelsea, 1; Everett, 2; Franklin, 1; Groton, 3; Medford, 1; Peabody, 1; Somerville, 2; Watertown, 1; Weymouth, 1; Worcester, 1; total, 33.

Undulant Fever: Brookline, 1.

OFFICERS OF THE MASSACHUSETTS MEDICAL SOCIETY, 1954–1955

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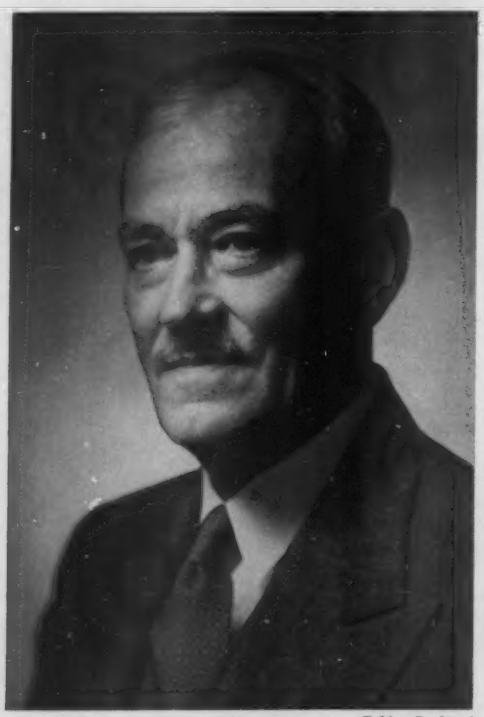

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ONE HUNDRED AND SEVENTY-FOURTH ANNIVERSARY OF THE MASSACHUSETTS MEDICAL SOCIETY

Tuesday, Wednesday and Thursday, May 17, 18 and 19

Hotel Statler, Boston

Committee on Nominations

The Committee on Nominations has presented the following slate to be voted upon by the Council at its annual meeting May 16, 1955:

President: Dr. Conrad Wesselhoeft, Boston
President-Elect: Dr. Harold R. Kurth, Lawrence
Vice-President: Dr. W. A. R. Chapin, Springfield
Secretary: Dr. Robert W. Buck, Waban
Treasurer: Dr. Eliot Hubbard, Jr., Cambridge
Assistant Treasurer: Dr. Norman A. Welch, West Roxbury
Orator: Dr. Merrill C. Sosman, Chestnut Hill

Delegates to the American Medical Association:
January 1, 1956—December 31, 1957
Dr. Norman A. Welch, West Roxbury
Dr. Harold R. Kurth, Lawrence
Dr. Nicholas S. Scarcello, Worcester

Alternates:
January 1, 1956—December 31, 1957
Dr. Carl Bearse, Cambridge
Dr. Robert W. Buck, Waban
Dr. Henry F. Howe, Cohasset

Alternate:
January 1, 1956—December 31, 1956
Dr. John F. Conlin, Winchester

Robert W. Buck, M.D., *Secretary*

Program

The Registration Desk will be located on the mezzanine floor, and all who attend the meeting are requested to register. The following events take place on the day prior to the official opening of the meeting.

MONDAY AFTERNOON, MAY 16

4:30 **Supervising Censors' Meeting** (Parlor B).

5:00 **Annual Meeting of the Council** (Georgian Room).

MONDAY EVENING, MAY 16

7:00 **Cotting Supper for Councilors** (Parlors A and B).

TUESDAY MORNING, MAY 17

First General Session

Georgian Room

9:00 *Motion picture* on automobile accidents entitled *"Search."* (From the Cornell University Automotive Safety Research Project.)

9:25 *Pulmonary Problems in the Newborn.* Dr. Virginia Apgar, professor of anesthesiology, Columbia University College of Physicians and Surgeons; attending anesthesiologist, Columbia-Presbyterian Medical Center, New York City.

9:50 *Clinical Dividends from Gastrointestinal Radiology.* Dr. Fred Jenner Hodges, professor of radiology, University of Michigan, Ann Arbor, Michigan.

10:15 **Recess.**

10:40 to 11:55 **Panel on Sympathectomy in Vascular Disease.** Moderator: Dr. Richard Warren, associate clinical professor of surgery, Harvard Medical School; senior associate in surgery, Peter Bent Brigham Hospital.
Dr. Edward A. Edwards, assistant clinical professor of anatomy, Harvard Medical School; associate in surgery, Peter Bent Brigham Hospital.
Dr. Karl Harpuder, assistant clinical professor of medicine, Columbia University College of Physicians and Surgeons; attending physician in charge of physical medicine and rehabilitation, Montefiore Hospital; senior consultant in peripheral vascular disease and physical medicine, Veterans Administration Hospital, New York City.
Dr. Robert R. Linton, assistant clinical professor of surgery, Harvard Medical School; visiting surgeon, Massachusetts General Hospital.
Dr. Reginald H. Smithwick, professor of surgery and chairman, Department of Surgery, Boston University School of Medicine; surgeon-in-chief, Massachusetts Memorial Hospitals.

TUESDAY NOON, MAY 17

Section Meetings and Luncheons

12:00 m. — 2:00 p.m.

Section of Medicine

Parlor A

Dr. Arthur S. Pier, Jr., Boston, *Chairman*
Dr. Ralph H. Wells, Lexington, *Vice-Chairman*
Dr. Harold F. Brown, Newton Centre, *Secretary*

Some Recent Advances in Knowledge of the Poliomyelitis Viruses. Dr. John F. Enders, associate professor of bacteriology and immunology, Harvard Medical School; chief, Research Division of Infectious Diseases, Children's Medical Center.

Section of Radiology

Parlor B

Dr. Robert E. Grandfield, Boston, *Chairman*
Dr. Martin H. Wittenborg, Boston, *Secretary*

Present Achievements and Future Promise of Radiology. Dr. Fred Jenner Hodges, professor of radiology, University of Michigan, Ann Arbor, Michigan.

Section of Physical Medicine

Parlor C

Dr. Fritz Friedland, Boston, *Chairman*
Dr. William J. Shriber, Boston, *Secretary*

Rehabilitation of the Patient with Arterial Disease of the Limbs. Dr. Karl Harpuder, assistant clinical professor of medicine, Columbia University College of Physicians and Surgeons; attending physician in charge of physical medicine and rehabilitation, Montefiore Hospital; senior consultant in peripheral vascular disease and physical medicine, Veterans Administration Hospital, New York City.

Section of Anesthesiology

Parlors D and E

Dr. M. Gene Black, Holyoke, *Chairman*
Dr. Bernard D. Briggs, Stoneham, *Secretary*

Obstetrics from the Anesthetist's Point of View. Dr. Virginia Apgar, professor of anesthesiology, Columbia University College of Physicians and Surgeons; attending anesthesiologist, Columbia-Presbyterian Medical Center, New York City.

TUESDAY AFTERNOON, MAY 17

2:30 **Annual Meeting of the Massachusetts Medical Society** (GEORGIAN ROOM).

Presentation of Awards to Medical Students

Presentation of Badges to Fifty-Year Members

Annual Discourse: *The Involuntary Plight of the Voluntary Hospital.* DR. CARL BEARSE, editor-in-chief, *Norfolk Medical News.*

WEDNESDAY MORNING, MAY 18

Second General Session

GEORGIAN ROOM

9:00 to 10:40 **Panel on Clinical and Epidemiologic Aspects of Poliomyelitis.** Moderator: DR. LOUIS WEINSTEIN, associate professor of medicine, Boston University School of Medicine; chief, Department of Infectious Disease, Massachusetts Memorial Hospitals.

DR. R. CANNON ELEY, assistant clinical professor of pediatrics, Harvard Medical School; chief, Isolation Service, Children's Medical Center.

DR. JOHANNES IPSEN, associate professor of public health, Harvard School of Public Health; superintendent, Institute of Laboratories, Massachusetts Department of Public Health.

DR. SIDNEY KIBRICK, instructor in pediatrics, Harvard Medical School; research associate, Research Division, Infectious Diseases, Children's Medical Center.

DR. CONRAD WESSELHOEFT, visiting lecturer in infectious diseases, Harvard Medical School.

11:05 THE SHATTUCK LECTURE. *The Portal Circulation.* DR. CHARLES G. CHILD, III, professor of surgery, Tufts College Medical School; director, First (Tufts) Surgical Service, Boston City Hospital.

WEDNESDAY NOON, MAY 18

Section Meetings and Luncheons

12:00 m. — 2:00 p.m.

Section of Obstetrics and Gynecology

PARLOR B

DR. JOHN R. BARKER, Needham, *Chairman*

DR. SMITH G. PHILIPS, Worcester, *Vice-Chairman*

DR. ARCHIE A. ABRAMS, Brookline, *Secretary*

Hemorrhagic Complications of Obstetrical Accidents. DR. MARIO STEFANINI, assistant professor of medicine, Tufts College Medical School; director of research and education, St. Elizabeth's Hospital.

Section of Pediatrics

PARLOR C

DR. FRED H. ALLEN, Holyoke, *Chairman*

DR. HARRY L. MUELLER, Winchester, *Secretary*

Antibiotics in Childhood. DR. HARRY SHWACHMAN, assistant professor of pediatrics, Harvard Medical School; chief, Division of Clinical Laboratories, Children's Medical Center.

Section of Physiology and Pathology

PARLORS D AND E

DR. DAVID SKINNER, Newton, *Chairman*

DR. JOHN D. HOUGHTON, Newton, *Vice-Chairman*

DR. HARRY G. OLKEN, Lynn, *Secretary*

Thyroiditis. DR. JOHN B. HAZARD, chief, Department of Pathology, Cleveland Clinic, Cleveland, Ohio.

Section of Industrial Health

PARLOR A

DR. KARL T. BENEDICT, West Boylston, *Chairman*

DR. THOMAS P. KENDRICK, Brookline, *Secretary*

Air Pollution as a Community Problem. PROFESSOR PHILIP DRINKER, professor of industrial hygiene, Department of Industrial Hygiene, Harvard School of Public Health.

WEDNESDAY AFTERNOON, MAY 18

Third General Session

GEORGIAN ROOM

2:00 to 3:15 **Panel on Rehabilitation and Workmen's Compensation.** Moderator: DR. DAVID HURWITZ, clinical associate in medicine, Harvard Medical School; chief, Division of Medicine, Mount Auburn Hospital; chief, Diabetes Service, Boston City Hospital.

DR. ALEXANDER P. AITKEN, clinical professor of orthopedic surgery, Tufts College Medical School; acting surgeon-in-chief, Sixth Surgical Service, Boston City Hospital; chairman, Subcommittee on Industrial Relations, American College of Surgeons.

DR. RICHARD J. CLARK, associate physician, Massachusetts General Hospital; director, Cardiac Work Classification Unit, Bay State Medical Rehabilitation Clinic; chairman, Rehabilitation Committee, Massachusetts Heart Association.

DR. HENRY H. KESSLER, clinical professor of rehabilitation, New York Medical College; medical director, Kessler Institute for Rehabilitation, West Orange, New Jersey.

MR. KENNETH E. POHLMANN, director, Rehabilitation for the Medical, Health and Hospital Services of the United Mine Workers of America Welfare and Retirement Fund; fellow, American Public Health Association; member, Board of Directors, National Rehabilitation Association; member, President's Committee on National Employment for the Physically Handicapped.

3:15 **Recess.**

3:30 to 5:00 **Panel on How to Make the Continuing Medical Education of Physicians Painless and Effective.** Moderator: DR. LAURENCE B. ELLIS, associate clinical professor of medicine, Harvard Medical School; president, Postgraduate Medical Institute; visiting physician and director, Cardiac Clinic, Boston City Hospital.

DR. JAMES M. FAULKNER, dean, Boston University School of Medicine.

DR. SAMUEL H. PROGER, professor of medicine and chairman, Department of Medicine, Tufts College Medical School; physician-in-chief, New England Center Hospital and Pratt Diagnostic Clinic.

DR. HENRY S. M. UHL, director of medical education and chief, Department of Education, Worcester City Hospital.

WEDNESDAY EVENING, MAY 18

6:00 - 7:00 **Cocktail Party** (PARLORS A, B and C).

7:00 **Annual Dinner of the Massachusetts Medical Society** (GEORGIAN ROOM).

Presiding: DR. CURTIS C. TRIPP, president, Massachusetts Medical Society.

Guest Speaker: His Excellency, CHRISTIAN A. HERTER, Governor of the Commonwealth of Massachusetts.

Ladies may be invited to attend the dinner.

Tickets for the cocktail party and dinner must be procured in advance.

THURSDAY MORNING, MAY 19

Fourth General Session

GEORGIAN ROOM

9:00 *Motion picture* entitled *"Nephrosis in Children."*

9:30-11:55 **Symposium on Pediatrics.** Staff of the Children's Medical Center.

THURSDAY NOON, MAY 19

Section Meetings and Luncheons

12:00 m. — 2:00 p.m.

Section of Surgery

PARLOR B

DR. CHESTER HOWE, Boston, *Chairman*

DR. FREDERICK P. ROSS, Fitchburg, *Secretary*

DR. WILLIAM W. BABSON, Gloucester, *Executive Committee*

DR. FREDERICK P. ROSS, Fitchburg, *Executive Committee*

DR. DOUGLAS A. FARMER, Boston, *Executive Committee*

Cancer of the Colon. DR. WARREN H. COLE, professor of surgery and head, Department of Surgery, University of Illinois College of Medicine.

Section of Dermatology

PARLORS D and E

DR. SALVATORE J. MESSINA, Newton Centre, *Chairman*

DR. WILLIAM R. HILL, JR., Boston, *Secretary*

The Differential Diagnosis of Dermatoses of the Hands. DR. JOHN G. DOWNING, professor of dermatology and syphilology emeritus, Tufts College Medical School and Boston University School of Medicine.

Section of Ophthalmology and Otolaryngology

PARLOR C

DR. BRENDAN D. LEAHEY, Lowell, *Chairman*

DR. EDGAR M. HOLMES, Auburndale, *Vice-Chairman*

DR. JOHN E. MCKEIGUE, Arlington, *Secretary*

Fractures of the Malar Bone with Orbital Deformity. DR. JOHN MARQUIS CONVERSE, associate professor of clinical surgery, New York University College of Medicine; visiting surgeon, Bellevue Hospital; surgeon director, Plastic Surgery Clinic, Manhattan Eye, Ear and Throat Hospital, New York City.

Section of Psychiatry and Neurology

PARLOR A

DR. HARRY C. SOLOMON, Boston, *Chairman*

DR. BARDWELL H. FLOWER, Worcester, *Secretary*

Problems of Work. DR. HENRY W. BROSIN, professor of psychiatry and chairman, Department of Psychiatry, University of Pittsburgh School of Medicine; director, Western Psychiatric Institute and Clinic, Pittsburgh, Pennsylvania.

THURSDAY AFTERNOON, MAY 19

Fifth General Session

GEORGIAN ROOM

2:00 *The Adverse Effects of Early Institutionalization of Mentally Subnormal Children.* DR. MALCOLM J. FARRELL, superintendent, Walter E. Fernald State School.

2:25 *The Doctor and His Patient.* DR. HENRY W. BROSIN, professor of psychiatry and chairman, Department of Psychiatry, University of Pittsburgh School of Medicine; director, Western Psychiatric Institute and Clinic, Pittsburgh, Pennsylvania.

2:50 *The Treatment of the Common Eruptions of the Hands.* DR. JOHN G. DOWNING, professor of dermatology and syphilology emeritus, Tufts College Medical School and Boston University.

3:15 **Recess.**

3:40 *Surgical Aspects of Jaundice.* DR. WARREN H. COLE, professor of surgery and head, Department of Surgery, University of Illinois College of Medicine.

4:05 *Surgical Treatment of Facial Injuries.* DR. JOHN MARQUIS CONVERSE, associate professor of clinical surgery, New York University College of Medicine; visiting surgeon, Bellevue Hospital; surgeon director, Plastic Surgery Clinic, Manhattan Eye, Ear and Throat Hospital, New York City.

4:30 *School Health and the Family Physician.* DR. KENNETH L. MACLACHLAN, chief of obstetrics, Melrose Hospital.

SCIENTIFIC EXHIBITS

BOOTH NO.

82 — JUVENILE RHEUMATOID ARTHRITIS (SPONDYLITIS).
Sponsor: Robert Breck Brigham Hospital.
Exhibitors: Dr. Theodore B. Bayles, Dr. Robert E. Barkin, Dr. Theodore A. Potter and Dr. J. Sydney Stillman.

83 — THE EXPIROGRAM IN PULMONARY AND CARDIAC DISEASE.
Sponsors: The Division of Clinical Immunology, Massachusetts Memorial Hospitals, and Boston University School of Medicine.
Exhibitors: Dr. William Franklin, Dr. Alan L. Michelson, Dr. Francis C. Lowell and Dr. Irving W. Schiller.

84 — GENERAL PRACTICE.
Sponsor: Massachusetts Academy of General Practice.

85 — REHABILITATION TECHNICS AND RESULTS.
Sponsor: Bay State Medical Rehabilitation Clinic.
Exhibitors: Dr. Arthur L. Watkins and staff of the Bay State Medical Rehabilitation Clinic.

86 — VOCATIONAL REHABILITATION.
Sponsor: Commonwealth of Massachusetts, Department of Education, Division of Vocational Rehabilitation.
Exhibitor: Division of Vocational Rehabilitation, Massachusetts Department of Education.

87 — BONE CHANGES IN THE BLOOD DYSCRASIAS.
Sponsors: New England Center Hospital and Tufts College Medical School.
Exhibitors: Dr. Arthur A. Thibodeau and Dr. Joseph K. Maloy.

88 — TREATMENT OF CARDIAC ARREST WITH AN EXTERNAL CARDIAC PACEMAKER.
Sponsor: Department of Medicine, Beth Israel Hospital.
Exhibitors: Dr. William Gibson, Dr. Arthur J. Linenthal, Dr. Leona R. Norman and Dr. Paul M. Zoll.

89 — AORTIC AND ILIAC COLLATERALS IN ARTERIOSCLEROSIS.
Sponsors: Department of Surgery, Peter Bent Brigham Hospital and Department of Radiology, Veterans Administration Hospitals, Boston and West Roxbury.
Exhibitors: Dr. Edward A. Edwards and Dr. Marjorie LeMay.

90 — CHRONIC RELAPSING PANCREATITIS.
Sponsor: The Lahey Clinic.
Exhibitors: Dr. Richard B. Cattell and Dr. Kenneth W. Warren.

91 — WHAT'S NEW AT THE BOSTON MEDICAL LIBRARY.
Sponsor: Boston Medical Library.

CORRIDOR—COMBINATION DRUG TREATMENT OF ESSENTIAL HYPERTENSION.
Sponsor: Department of Medicine, Massachusetts Memorial Hospitals.
Exhibitors: Dr. Robert W. Wilkins, Dr. Walter E. Judson and Dr. William Hollander.

CORRIDOR—CANCER RESEARCH.
Sponsor: Cancer Research Institute of the New England Deaconess Hospital.
Exhibitors: Dr. Shields Warren and staff of the Cancer Research Institute of the New England Deaconess Hospital.

TECHNICAL EXHIBITORS

1955

	BOOTH NO.
Abbott Laboratories	41 & 42
The Alkalol Company	25
American Hospital Supply Corporation	70

Ames Company, Inc. 78
Ayerst Laboratories 24
Baby Development Clinic 5
Chester A. Baker Laboratories, Inc. 19
The Baker Laboratories, Inc. 35
Baxter Laboratories, Inc. 70
Beech-Nut Packing Company 62
Bilhuber-Knoll Corporation 47
The Borden Company 75
Brewer & Company, Inc. 73
Brown and Connolly, Inc. ..... Stage B
Buffington's, Inc. 55
Burroughs Wellcome & Company, Inc. 27
Cambridge Instrument Company, Inc. 7
S. H. Camp and Company 36
Cardiotron (Samuel Mushlin) 8-A
Carnation Company 13
Ciba Pharmaceutical Products, Inc. ..... 31 & 32
The Coca-Cola Company 38
Coles Electronic Corporation 3
Crosbie-Macdonald 49
Davies, Rose & Company, Ltd. 44
Desitin Chemical Company 10
Doho Chemical Corporation 39
Dy-Dee Service, Inc. 12
Endo Products, Inc. 52
Charles O. Finley & Company 6
C. B. Fleet Company, Inc. 66
Geigy Pharmaceuticals 59
Gerber Products Company 71
Lawrence E. Gott 64
Gray Pharmaceutical Company, Inc. 17
H. J. Heinz Company 45
Hoffmann-La Roche, Inc. 20
H. P. Hood & Sons 58
Hornblower & Weeks 15
Ives-Cameron Company 63
George Laben 29
Lederle Laboratories 26
Eli Lilly and Company 33
P. Lorillard Company, Inc. 14
E. F. Mahady Company 16
Maltex Company ..... Stage A
M & R Laboratories 68
Massachusetts Medical Service 11
McNeil Laboratories, Inc. 51
Mead Johnson & Company 28
Medical Clearing Bureau, Inc. 7-A
The Medical Protective Company 30
The Wm. S. Merrell Company 53
The National Drug Company 4
Nepera Chemical Company, Inc. 1
The Nestlé Company, Inc. 37
New England Dairy and Food Council 2
Parke, Davis and Company 76
The E. L. Patch Company 22
Pet Milk Company 67
Pfizer Laboratories 79
Pitman-Moore Company 23
Postgraduate Medical Institute 9
Professional and Hospital Equipment Company 61
The Purdue Frederick Company 64-A
R. J. Reynolds Tobacco Company 54
Riker Laboratories, Inc. 18
A. H. Robins Company, Inc. 50
J. B. Roerig and Company ..... Stage C
Sanborn Company 65
Sandoz Pharmaceuticals 40
Schering Corporation 74
G. D. Searle & Company 72
Sharp & Dohme 81
Carroll Dunham Smith Pharmacal Company 69
Smith, Kline & French Laboratories 46
Smith's Photographic Store 1-A
E. R. Squibb & Sons 48
Surgeons' and Physicians' Supply Company 77
Swift & Company 60
Tailby-Nason Company 21
U. S. Vitamin Corporation 8
The Upjohn Company 80
Varick Pharmacal Company, Inc. 56
Walker Laboratories, Inc. 92
Warner-Chilcott Laboratories 57
White Laboratories, Inc. 34
Winthrop-Stearns, Inc. 43

DINNER FOR THE PRESIDENTS OF THE DISTRICT MEDICAL SOCIETIES

TUESDAY EVENING, MAY 17

PARLOR C

6:30 p.m.

ANNUAL GOLF TOURNAMENT

WEDNESDAY AFTERNOON, MAY 18

1:00 p.m.

All members and guests are welcome.

(Golf Course to be announced later)

THE MASSACHUSETTS MEDICO-LEGAL SOCIETY

ANNUAL MEETING

WEDNESDAY AFTERNOON, MAY 18

PARLOR C

2:30 p.m.

EXHIBITION OF WORKS OF ART

BY MEMBERS OF THE MASSACHUSETTS PHYSICIANS ART SOCIETY

FOURTH FLOOR

Daily: 9:00 a.m. — 5:00 p.m.

MAY 17, 18 and 19

AMERICAN MEDICAL ASSOCIATION

CLINICAL SESSION IN BOSTON

NOVEMBER 29, 30, DECEMBER 1, 2

MECHANICS BUILDING (HEADQUARTERS: HOTEL STATLER)

DR. FRANK P. FOSTER, *General Chairman*

DR. LAURENCE B. ELLIS, *Co-Chairman*

DR. THEODORE L. BADGER

Chairman, Program Committee

WOMAN'S AUXILIARY TO THE MASSACHUSETTS MEDICAL SOCIETY

The registration desk for the meeting on May 17 will be in the lobby, Hotel Vendome, with registration from 9:00 a.m. to 4:00 p.m., and the registration desk for the meeting on May 18 will be in the mezzanine, Hotel Statler, with registration from 9:00 a.m. to 3:00 p.m.

TUESDAY MORNING, MAY 17

10:00 **Annual Meeting** (SALON, FIRST FLOOR, HOTEL VENDOME).

TUESDAY AFTERNOON, MAY 17

12:45 **Luncheon** (EMPIRE ROOM, FIRST FLOOR, HOTEL VENDOME; $2.88, including Tax and Tip).

2:15 **Afternoon Session** (SALON, HOTEL VENDOME).

3:30 **Executive Board Meeting** (MARLIN ROOM, HOTEL VENDOME).

3:45 **Sherry Hour** (EMPIRE ROOM, HOTEL VENDOME).

WEDNESDAY MORNING, MAY 18

10:30 **Bus Tour Starting from Columbus Avenue Entrance of the Hotel Statler for Harvard Yard, Cambridge.** (Transportation provided.)

11:00 **Tour of Harvard Yard.** (Buses leave for the Hotel Continental, Cambridge, at 12:30 p.m.)

1:15 **Luncheon** (Hotel Continental, Cambridge; $3.00, including Tax and Tip).

WEDNESDAY EVENING, MAY 18

6:00 - 7:00 **Cocktail Party** (with members of the Massachusetts Medical Society, Hotel Statler).

7:00 **Dinner** (with members of the Massachusetts Medical Society, Hotel Statler).

MASSACHUSETTS MEDICAL SOCIETY

COMMITTEE ON MATERNAL WELFARE

(JOHN F. JEWETT, M.D., CHAIRMAN)

Minimum Standards of Obstetric Care

During 1954 in Massachusetts 55 mothers died in childbirth, with over 95,000 babies born. The statistics, which will be published in the annual report of the Committee on Maternal Welfare, continue the prevailing trend in distribution of preventability — that is, about only a third were nonpreventable. Regardless of what the *rate* may appear to be, the remaining women should not have died, and it is clear that the medical profession has a grave responsibility to eliminate needless deaths in the future. However, no one physician or group of physicians, particularly sitting in closed session around the conference table, can tell another how he should practice any phase of medicine under a given set of circumstances. On the other hand, careful analysis of past deaths reveals that simple adherence to well established and freely accepted principles as taught in every medical school, combined with a certain minimum of equipment and facilities, would have avoided the majority of them. Today, "minimum facilities" must include immediately available blood for transfusion.

Accordingly, the Committee on Maternal Welfare has drawn up a set of "Minimum Standards of Obstetric Care" for the information and, it is hoped, guidance of all concerned. It is emphasized that these do not represent in any sense the *optimum* or in fact even "good obstetrics," but the intent has been so to design them that no patient will die a "preventable death" if each of these standards is met.

Prenatal Care

Blood pressure, weight and *urinary albumin* must be recorded at every visit.

Hemoglobin must be recorded at the first visit, and repeated at least in the seventh month on all patients. In those requiring treatment for anemia it must be repeated again and again.

Blood must be tested for syphilis and for blood grouping and Rh factor at the first visit.

Visits must be required of all patients at least monthly in the first six months, every two weeks during the seventh and eighth months and every week in the ninth month of pregnancy; in case of complications, especially toxemia, weekly visits begin earlier (often, patients must be seen two or three times in a week).

History must be obtained at first visit regarding previous pregnancies, menstruation (including date of last menstrual period and a carefully estimated date of confinement) and nephritis or any kidney ailment, hypertension, diabetes, scarlet fever, rheumatic fever or any form of heart disease.

Measurements of the pelvis must be made, including at least the diagonal conjugate and a realistic appraisal of the outlet.

Heart, abdomen and *pelvis* must also be carefully examined, and the urine for sugar.

Intra-partum and Post-partum Care

On Admission

The nurse must note the length of gestation, temperature, pulse and respirations, blood pressure and fetal heart rate and immediately report if any is abnormal. In active labor determination of the blood pressure and pulse is repeated at least hourly.

The activity of labor, presence and amount of bloody show and state of membranes must be recorded.

The time of last meal or fluid must be called to the attention of the doctor.

During Labor

No street clothes should be worn in the labor room — caps and masks are mandatory when the perineum is exposed.

Constant attendance by a nurse is necessary. One nurse may attend two or more patients if they are in the same room. A nurse must be literally at the bedside of all medicated patients; attendance at a desk in the room is permissible with unmedicated patients.

A doctor must be on the obstetric floor when a primigravida is fully dilated and a multipara is 4 or 5 cm. ("3 fingers") dilated. Medication by barbiturates, narcotics, scopolamine and adjuvant drugs always requires a doctor's order and should be on an individual basis. Use of analgesics must be limited when nursing personnel is not adequate for bedside attendance.

Pituitary extract should not be given except on individual order and with extreme caution.

No vaginal examination should be made with a patient in labor except under sterile conditions.

No rectal or vaginal examination should be made on a patient who is bleeding unless in the operating room with a team prepared for instantaneous cesarean section.

Precipitate Delivery

If precipitate delivery threatens the head must not be held back by any method. Delivery of the head may be controlled by the attendant but never delayed. If uncontrolled precipitate delivery occurs the blood pressure and pulse must be checked immediately and every five minutes for half an hour, the fundus held, and the patient watched carefully for bleeding from hidden lacerations.

Anesthetic

The anesthetic must be given only by a qualified person and never by the circulating nurse.

Inhalation anesthetic must never be used unless the patient's stomach is empty. Spinal or caudal anesthetic must be given only by a doctor familiar with its risks who will take time to stabilize the patient before proceeding. (Stabilization refers particularly to a fall in blood pressure that can be corrected with pressor drugs and to the level of anesthesia, which must not ascend above the costal margin.)

The anesthetist must be in constant personal attendance at the patient's head throughout the entire obstetric or surgical procedure and is responsible for her further care until she has recovered from anesthesia.

Delivery

The absolute minimum personnel includes obstetrician, anesthetist and circulating nurse. Added nursing personnel should be within a moment's call for unforeseen emergencies.

Delivery through an undilated cervix is extremely dangerous. High-forceps delivery is not justified today. Excessive fundal pressure is dangerous.

A consultant or assistant should be called in cases entailing more than low-forceps delivery.

Asepsis must be surgically strict. Masks must cover noses.

Excessive Credé maneuver is dangerous.

Post Partum (the hazard of hemorrhage is highest now)

Even if oxytocics are used for the third stage it is imperative to guard the fundus immediately after delivery of the baby until forty-five minutes after expulsion of the

placenta (this means holding the fundus through the abdomen between two hands, presumably by a nurse).

Cross matching of blood is started immediately if postpartum bleeding exceeds more than a slight trickle.

If in doubt the attendant should start the transfusion and call for help.

Ample supplies of compatible blood must be readily available in the hospital, preferably on the delivery floor, twenty-four hours a day.

The vagina should never be packed for bleeding from above.

The cervix is explored visually, and the vagina manually, for lacerations if bleeding occurs.

If bleeding persists immediate assistance is essential.

The standards are the result of extremely careful and prolonged study by the entire committee. They have been put in the briefest possible form for the sake of emphasis and clarity. They contain nothing new or revolutionary. Their inadequacy as a standard of excellence is recognized, but for every heading in this set there is on record a maternal death that could have been avoided if the dictum had been observed. It is re-emphasized that these are set forth with the purpose of avoiding deaths; the Committee considers this perhaps its prime responsibility.

CIVIL DEFENSE AGENCY

THE NATURE AND SIGNIFICANCE OF RADIOACTIVE FALLOUT

With the detonation of a thermonuclear bomb such as the hydrogen bomb, four major effects are to be noted: blast, heat, instantaneous nuclear radiation and residual radioactivity. The first three occur at the time of detonation. With a 35-megaton hydrogen bomb immediate nuclear radiation does not present a serious hazard beyond a radius of approximately 18 miles from ground zero, where heat and blast are of great concern. The area of blast, heat and nuclear radiation varies according to the energy liberated at the time of explosion. In general an ordinary 1X atomic bomb is equivalent to 20,000, and the 35-megaton hydrogen bomb to 35,000,000 tons of TNT.

Atomic detonations may be divided into two main types:

Air bursts detonated 2000 feet or more above the ground in which the fireball (an intensely luminous core that develops in the surface winds) does not intersect the ground.

Surface bursts or low air bursts (within 500 feet of the ground) in which the fireball intersects the ground.

Outside the area of blast and immediate radiation damage from atomic explosion, a hazard exists from fallout of radioactive particles created by the explosion. Fallout is composed of fission products, neutron-induced radioactivity in elements of earth or water, and uranium 235 and plutonium 239 that have escaped fission. In air burst the radioactive débris consists of the remains of the bomb and casing and other equipment utilized in the explosion that has been vaporized. The amount of particles occurring in air bursts is relatively small, and the particles are of small dimension so that in the absence of rainfall or snow, the hazard from this type of explosion beyond the area of blast and thermal damage is slight.

In a surface burst thousands of tons of soil are carried into the atmosphere in the bomb cloud. These particles, which are made radioactive by neutrons liberated at the time of the explosion itself, serve as condensation surfaces for the vaporized material. The particles are much larger than those in an air burst and, therefore, precipitate more quickly. In general, fallout is governed by the direction and velocities of prevailing surface winds, which, in Massachusetts, are southwesterly at a speed of 12 to 15 miles per hour. It should be noted, however, that crosscurrents in the upper atmosphere and base of the stratosphere between levels of 30,000 and 50,000 feet may cause deviation in the general pattern of direction of fallout. The radioactivity of fallout decreases rapidly with time.

In March, 1954, a thermonuclear bomb was exploded at Bikini Atoll, on a coral island. Coral consists mainly of calcium carbonate, and in this explosion great quantities of particles composed mostly of slaked lime were taken into the atmosphere. Those particles ranged from 1/1000 to 1/50 of an inch in diameter, were white, resembling snow, and were somewhat adhesive. The large cloud formed by the explosion moved in the direction of the prevailing winds, which were westerly, and the radioactive particles were deposited in the direction of the wind over an elliptical or cigar-shaped area approximately 220 miles long and varying in width from 20 to 40 miles. The fallout began to occur approximately eight hours after the explosion and continued for several hours.

The contamination from radioactive fallout may last from eight hours up to several years, depending upon the radioactive half life of the elements involved. Any total general body exposure in excess of 600 r is considered lethal. Approximately 30 r of radioactivity received in a brief period will cause temporary changes in the blood. The roentgen is the fundamental unit of dosage for x-rays and gamma radiation and represents 83 ergs of absorbed energy per gram of air. Irradiation in general affects the body by causing ionization in the individual cells, with breaking up of the chromosomes, swelling of the nucleus and of the entire cell and ultimate destruction of the cell. Effects of exposure to radiation limited to certain parts of the body are called radiation injuries and consist of local necrosis, alopecia and sterility. Effects of exposure of the entire body that are systemic rather than local constitute radiation illness. The biologic effects of the different types of radiation on living organisms are similar but differ in intensity, depending on the total amount absorbed, the rate of absorption (acute or chronic dosage) and the area of body exposed to the

radiation. Thus, a total dose of 600 r would be fatal if absorbed in one day by the whole body, but would probably have a negligible effect if absorbed over a period of thirty years (Table 1).

TABLE 1. *Effects of Acute Radiation Dosage upon the Whole Body.*

DOSAGE r	EFFECT
0 - 25	No obvious injury
25 - 50	Possible blood changes but no serious injury
50 - 100	Blood-cell changes; some injury; no disability.
100 - 200	Injury; possible disability.
200 - 400	Injury & disability certain; death possible.
400	Fatal to 50% (median lethal dose); nausea, vomiting, malaise, fever, diarrhea & tendency to hemorrhages; latent period, several days to 2 wk.; death 2-12 wk. after exposure.
600	Fatal lethal dose; death usual within 2 wk. of exposure.
Several thousand (extreme dose)	Death within few hours

Simple precautionary measures can greatly reduce the hazard to life from alpha, beta and gamma radiation that ensues from radioactive fallout. One can reduce the exposure by taking shelter within a house or basement or cellar and by simple decontamination steps. It has been found that the radiation which ensued in the first floor of an ordinary frame house in an area of fallout was about half the level that occurred outside. Much greater protection would result in the basement of a brick or stone house. In an old type of cyclone cellar, with a covering of approximately 3 feet of earth, the radiation level would be reduced to 1/5000 or to a level completely safe even in the most heavily contaminated areas.

Radioactive material that precipitates during a fallout may or may not be visible but can be detected by such an instrument as a Geiger counter or an ionization chamber. However, any dust or ash that is carried downwind a few hours after a thermonuclear explosion should be considered radioactive unless measured by a radiation instrument and found to be harmless.

When fallout particles come into contact with skin, hair or clothing, decontamination measures should be taken immediately, the most effective and simple being thorough bathing of the exposed parts of the body and a change of clothing. Precautions should be taken to avoid the use of foods and liquid that may contain fallout particles. Fresh food suspected of being contaminated should be destroyed. Open water supplies, such as reservoirs, that have become contaminated by radioactive fallout must be checked before use. Deep wells or other underground sources of water can be safely used.

The two isotopes — radiostrontium and radioactive iodine — constitute the two principal hazards from radioactivity produced by detonations of atomic weapons. Radioactive iodine has a relatively short half life of eleven and a half days, and the possibility of its causing serious hazards is limited. Radiostrontium, on the other hand, has a very long half life of approximately thirty years. The amount of radiostrontium that would result from nuclear explosions is exceedingly small.

In conclusion general protective measures that can be taken against radioactive fallout are shelter and decontamination, and the avoidance of ingestion or inhalation of contaminated mediums from the environment into the body. Burns, hematologic and bone-marrow changes and infection, being part of the general radiation syndrome, will also be encountered in persons who have been exposed to the hazard of radioactive fallout.

The Commonwealth of Massachusetts has 6 teams of trained radiologic monitoring personnel equipped with survey meters located at Amherst, Pittsfield, Cambridge, Reading, Newton and Quincy, and approximately 200 trained radiologic monitors are available. Manuals outlining the organization and operation of monitoring programs have been prepared by the Medical Service of the Massachusetts Civil Defense Agency. A training manual on the nature and scope of atomic radiation and the use of radiation-detection instruments is also available for physicians.

THOMAS F. MAHER, M.D., D.P.H.
Executive Medical Officer

BIBLIOGRAPHY

Cronkite, E. P., and Chapman, W. H. Critical analysis of syndrome of acute total body radiation illness, its role in atomic warfare and its influence on future practice of military medicine. *Mil. Surgeon* **104**: 7-21, 1949.

Evans, E. I. Burn problem in atomic warfare. *J.A.M.A.* **143**:1143-1146, 1950.

United States War Department. *United States Strategic Bombing Survey: Effects of atomic bombs on health and medical services in Hiroshima and Nagasaki.* 91 pp. Washington, D. C.: Government Printing Office, 1947.

Warren, S. Effects of radiation on normal tissues. *Arch. Path.* **34**:443-450, 562-608, 749-787, 917-931 and 1070-1084, 1942.

NOTICES

HARVARD MEDICAL SOCIETY

The Harvard Medical Society will present the undergraduate assembly of Harvard Medical School in the auditorium, Jimmy Fund Building, 35 Binney Street, Boston, on Tuesday, May 10, at 7:30 p.m.

The program is as follows:

Effect of Right Auricular Pressure upon Intrinsic Cardiac Rhythmicity. John R. Blinks, IV Year.

Experimental Closure of Ventricular Septal Defects. Judah Folkman, II Year.

Nonsteroid Ovarian Hormones. Bernard Kliman, IV Year.

Inhibition of Polymorphonuclear Leukocyte Phagocytosis by Heterologous Antibodies. Robert H. Resnick, IV Year.

Cellular Localization of Boron-10 and Lithium-6 by Means of Neutron-Capture Autoradiography. Leon C. Edwards, IV Year.

Quantitative Changes in Blood Flow Following Experimental Denervation of the Hind Limbs. Russell A. Rohde, III Year.

Some Effects of Pilocarpine and Atropine on the Dog Heart-Lung Preparation. Leonard J. Friedman, IV Year.

Effect of Alloxan Diabetes on Caries Incidence in the Albino Rat. Martin S. Nichols, IV Year.

Presentation of the Soma Weiss Award by the Faculty Committee.

NEW ENGLAND PEDIATRIC SOCIETY

The spring meeting of the New England Pediatric Society will be held (day sessions starting at 11 a.m.) in Fitken Amphitheater, New Haven Hospital, New Haven, and (starting at 5 p.m.) at the New Haven Medical Association, 364 Whitney Avenue, New Haven, on Wednesday, May 11. Dr. Myron Wegman will speak on the topic "Pediatrics in the World Health Organization" at the evening meeting.

ALPHA OMEGA ALPHA LECTURE

The annual spring lecture sponsored by Alpha Omega Alpha Society of Boston University School of Medicine will be delivered by Dr. Gustaf E. Lindskog, Yale University School of Medicine, who will speak on the topic "Diseases of the Diaphragm," in the auditorium, Boston University School of Medicine, on Monday, May 2 at 5 p.m. All interested persons are invited.

BENJAMIN WATERHOUSE MEDICAL HISTORY SOCIETY

The Benjamin Waterhouse Medical History Society, recently organized at Boston University School of Medicine, will hold its third monthly supper meeting at 6:00 p.m., Tuesday, May 3 in the Musket Room of the Howard Johnson Restaurant at Southampton Street and Massachusetts Avenue. Dr. Lamar Soutter will talk on the surgical processes during the life and times of Dr. Waterhouse.

At the first meeting, in February, Dr. John J. Byrne, president of the Society, talked on Benjamin Waterhouse. At the second meeting, on March 28, Dr. Earl H. Dearborn presented a paper on "Therapeutic Agents of the 18th and 19th Centuries."

The secretary of the Society is Dr. David McL. Greeley, 818 Harrison Avenue, Boston 18.

EUROPEAN RHEUMATOLOGY CONGRESS

The third European Rheumatology Congress will be held in Scheveningen, The Hague, June 13-17.

A day each will be devoted to the topics "Rheumatism and Social Medicine," "Connective Tissue" and "Osteoarthritis of the Spine and Disk Degeneration" and "Rheumatic Fever."

Applications for membership should be submitted to the Secretariate: Burg. de Monchyplein 9, The Hague, before May 1. The fees will include preprints, congress proceedings and social events.

SOCIETY MEETINGS AND CONFERENCES

May. Sanitary Engineering Center Training Programs. Page 122, issue of July 15.

April 28–May 26. Boston City Hospital. House Officers' Lectures. Page 509, issue of March 24.

April 28–30. American Goiter Association. Page 329, issue of February 24.

April 30. State University College of Medicine at New York City. Page 289, issue of February 17.

April 30. Phi Delta Epsilon Fraternity. Graduate Club of Boston. Page 461, issue of March 17.

May 2. Alpha Omega Alpha Lecture. Notice above.

May 2–31. Consultation Clinics for Crippled Children in Massachusetts. Page 733.

May 3. Benjamin Waterhouse Medical History Society. Notice above.

May 4 and 5. American Psychosomatic Society. Page 286, issue of August 12.

May 4–25. Lahey Clinic Lectures. Page 289, issue of March 17.

May 4–June 15. Boston State Hospital. Psychiatry Seminars. Page 461, issue of March 17.

May 6–8. Student American Medical Association. Page 461, issue of March 17.

May 7. Middlesex South District Medical Society. Postgraduate Course. Page 758, issue of October 28.

May 10. Harvard Medical Society. Page 741.

May 11. New England Pediatric Society. Notice above.

May 13 and 14. American Association for Cleft Palate Rehabilitation. Page 461, issue of March 17.

May 13–15. American Academy of Dental Medicine. Page 354, issue of March 31.

May 17–19. Massachusetts Medical Society. Annual Meeting. Hotel Statler, Boston.

May 18–20. American College of Cardiology. Page 414, issue of March 10.

May 23–27. American Trudeau Society. Page 462, issue of March 17.

May 29–June 2. American Society of X-Ray Technicians. Page 553, issue of March 31.

June 1–4. American Proctologic Society. Page 648, issue of April 14.

June 1–5. American College of Chest Physicians. Page 553, issue of March 31.

June 5 and 6. American Board of Physical Medicine and Rehabilitation. Page 794, issue of November 4.

June 6–10. Woman's Auxiliary to the American Medical Association. Page 647, issue of April 14.

June 7–9. United States Public Health Service Medical-Officer Examinations. Page 509, issue of March 24.

June 10–12. American Electroencephalographic Society. Page 647, issue of April 14.

June 13–15. American Neurological Association. Page 689, issue of April 21.

June 13–17. European Rheumatology Congress. Notice above.

June 21–25. National Association for the Prevention of Tuberculosis. Page 540, issue of March 25.

July, August and September. Beth Israel Hospital Course. Page 509, issue of March 24.

July 10–16. Congress of Nobel Prize Winners. Page 510, issue of March 24.

July 11–15. Symposium on Tuberculosis. Page 509, issue of March 24.

August 28–September 2. American Congress of Physical Medicine and Rehabilitation. Page 157, issue of January 27.

October 6–8. Academy of Psychosomatic Medicine. Page 509, issue of March 24.

October 22–26. American Heart Association. Scientific Session. Page 603, issue of April 7.

October 25–27. American Institute of Dental Medicine. Page 414, issue of March 10.

November 6–13. Second International Congress of Allergology. Page 157, issue of January 27.

November 19–21. American Association of Blood Banks. 8th Annual Meeting. Palmer House, Chicago, Illinois.

November 29–December 2. Interim Session of the American Medical Association. Boston.

Calendar for the Week Beginning Thursday, May 5

Thursday, May 5

*8:00–8:45 a.m. Case Presentations. Joslin Clinic. Joslin Auditorium, New England Deaconess Hospital.
*8:45–9:45 a.m. Cardiac Grand Rounds. Jimmy Fund Building, Children's Medical Center, 35 Binney Street.
*9:00–10:00 a.m. Surgical Rounds. Sherman Auditorium, Beth Israel Hospital.
*9:00–10:00 a.m. Arthritis Grand Rounds. Robert Breck Brigham Hospital.
*10:00–11:00 a.m. Combined Medical-Surgical Rounds. Sherman Auditorium, Beth Israel Hospital.
10:30–11:15 a.m. Lecture on Diabetes for Doctors and Patients by a Member of the Joslin Clinic. Joslin Auditorium, New England Deaconess Hospital.
*11:00 a.m.–12:00 m. Thoracic Conference. Embryology of the Heart. Dr. Dwight E. Harken. Main Amphitheater, Peter Bent Brigham Hospital.
*11:00 a.m.–12:00 m. Medical Staff Conference. Sherman Auditorium, Beth Israel Hospital.
*11:00 a.m.–12:30 p.m. Hand Clinic (Physical Medicine and Rehabilitation Service). Boston City Hospital.
*12:00 m.–1:00 p.m. Surgical Staff Conference. Sherman Auditorium, Beth Israel Hospital.
*12:15–1:15 p.m. Orthopedic Clinic. Amphitheater, Peter Bent Brigham Hospital.
*4:00–5:00 p.m. Pediatric Seminar. Burnham Memorial Hospital for Children (Burnham 4), Massachusetts General Hospital.

Friday, May 6

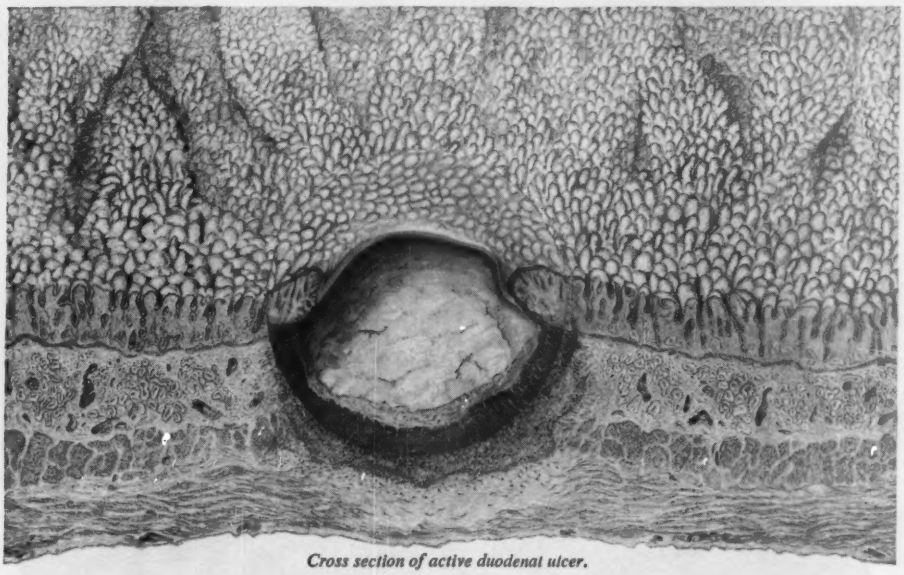
*8:00–8:45 a.m. Case Presentations. Joslin Clinic. Joslin Auditorium, New England Deaconess Hospital.
*9:30–11:30 a.m. Medical and Surgical Grand Rounds. Drs. George W. Thorn and Francis D. Moore. Main Amphitheater, Peter Bent Brigham Hospital.
*10:00 a.m. Tuberculosis Surgical Clinic. South End Health Unit, 57 East Concord Street.
10:30–11:15 a.m. Lecture on Diabetes for Doctors and Patients by a Member of the Joslin Clinic. Joslin Auditorium, New England Deaconess Hospital.
*11:00 a.m.–1:00 p.m. Medical Grand Rounds. Dr. Proger and Staff. Stearns Auditorium, New England Center Hospital.
*1:30 p.m. Fertility and Endocrine Clinic. Free Hospital for Women, Brookline.
*1:30 p.m. Tumor Clinic. Mount Auburn Hospital, Cambridge.
*4:00–5:00 p.m. Child Psychiatry. Burnham Memorial Hospital for Children (Burnham 4), Massachusetts General Hospital.
*4:00–10:00 p.m. Alcoholism Clinic. By appointment. Washingtonian Hospital, 41 Morton Street, Jamaica Plain.

Saturday, May 7

*8:00–8:45 a.m. Case Presentations. Joslin Clinic. Joslin Auditorium, New England Deaconess Hospital.
*8:00–9:00 a.m. Anesthesia Conference. Dr. Etsten. Pratt Lecture Hall, Joseph H. Pratt Diagnostic Hospital.
*8:30–10:00 a.m. Orthopedic Staff Conference. Boston City Hospital.
*9:00–10:30 a.m. Surgical Grand Rounds. Dr. Child and Staff. Stearns Auditorium, New England Center Hospital.

(Concluded on page xxvi)

PRO-BANTHĪNE® IN DUODENAL ULCER



Cross section of active duodenal ulcer.

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1. Ruffin, J. M.; Baylin, G. J.; Legerton, C. W., Jr., and Texter, E. C., Jr.: Mechanism of Pain in Peptic Ulcer, Gastroenterology *23*:252 (Feb.) 1953.

2. Schwartz, I. R.; Lehman, E.; Ostrove, R., and Seibel, J. M.: A Clinical Evaluation of a New Anticholinergic Drug, Pro-Banthine, Gastroenterology *25*:416 (Nov.) 1953.

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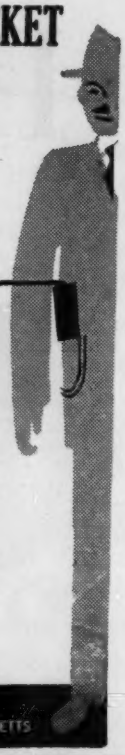

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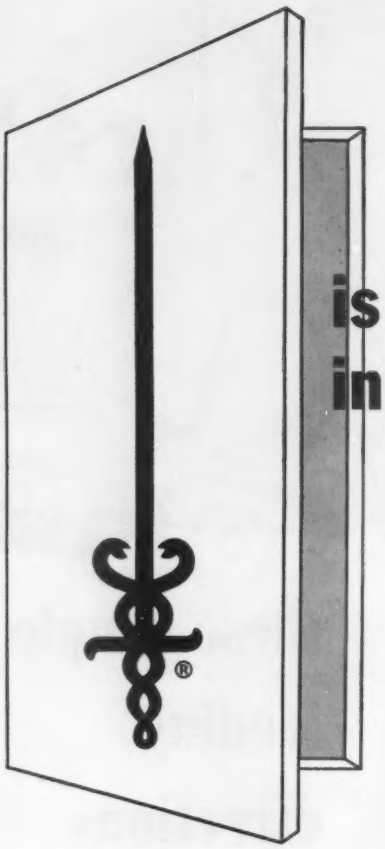

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10:30–11:15 a.m. Lecture on Diabetes for Doctors and Patients by a Member of the Joslin Clinic. Joslin Auditorium, New England Deaconess Hospital.
10:30 a.m.–12:00 m. Middlesex South District Medical Society. Postgraduate Course. Office Management of Minor Surgery and Rectal Diseases. Drs. Thomas J. Anglem, Edwin P. Hayden, Roy E. Mabrey and Moderator, Dr. Gaspar Angelo. Margaret Jewett Hall, Mount Auburn Hospital, Cambridge.
*11:00 a.m.–12:00 m. Hematology Clinic. Dr. William Dameshek. Pratt Lecture Hall, Joseph H. Pratt Diagnostic Hospital.

MONDAY, MAY 9
*8:00–8:45 a.m. Case Presentations. Joslin Clinic. Joslin Auditorium, New England Deaconess Hospital.
*8:00–9:00 a.m. Medical and Surgical Clinic on Diabetic Problems. New England Deaconess Hospital.
*8:30–9:30 a.m. Clinic by Entire Surgical Staff and Anesthesia Staff. New Cheever Amphitheater, Dowling Building, Boston City Hospital.
10:30–11:15 a.m. Lecture on Diabetes for Doctors and Patients by a Member of the Joslin Clinic. Joslin Auditorium, New England Deaconess Hospital.
*12:00 m.–1:00 p.m. Therapeutic Conference. Dr. McCombs. Stearns Auditorium, New England Center Hospital.
*12:15–1:15 p.m. Clinicopathological Conference. Main Amphitheater, Peter Bent Brigham Hospital.
*1:00 p.m. Alcoholism Clinic. Peter Bent Brigham Hospital.
*4:00 p.m. Clinicopathological Conference. Pathology Conference Room, Beth Israel Hospital.
*4:00–5:00 p.m. Surgical Pathology. Dr. MacMahon. Stearns Auditorium, New England Center Hospital.
*4:00–5:30 p.m. Anesthesiology Conference. Case Reports. Dr. Burnap. Surgical Conference Room, Peter Bent Brigham Hospital.
*5:00–6:30 p.m. Anesthesia Group of the Lahey Clinic. Joslin Auditorium, New England Deaconess Hospital.
*7:00–9:00 p.m. Slide Seminar in Pathology. Dr. MacMahon. Stearns Auditorium, New England Center Hospital.

TUESDAY, MAY 10
*8:00–8:45 a.m. Case Presentations. Joslin Clinic. Joslin Auditorium, New England Deaconess Hospital.
*9:00 a.m. Geriatrics Clinic. Peter Bent Brigham Hospital.
10:30–11:15 a.m. Lecture on Diabetes for Doctors and Patients by a Member of the Joslin Clinic. Joslin Auditorium, New England Deaconess Hospital.
*12:00 m. Pediatric Grand Rounds. New Cheever Amphitheater, Dowling Building, Boston City Hospital.
*12:00 m.–1:00 p.m. Endocrine Conference. Dr. Astwood. Stearns Auditorium, New England Center Hospital.
*12:00 m.–1:00 p.m. Pediatric Grand Rounds. Burnham Memorial Hospital for Children (Burnham 4), Massachusetts General Hospital.
*12:15–1:15 p.m. X-Ray Conference. Dr. Merrill Sosman. Main Amphitheater, Peter Bent Brigham Hospital.
12:30–1:30 p.m. Medical Journal Club. Joslin Clinic, New England Deaconess Hospital.
*4:30–6:00 p.m. Anesthesia Conference. Dr. Etsten. Stearns Auditorium, New England Center Hospital.
*5:00 p.m. Surgical Journal Review. Mount Auburn Hospital, Cambridge.
*5:00–6:00 p.m. Service Meeting followed by Clinical Conference (Medical, Surgical, Obstetrical). Faulkner Hospital.
*7:00 p.m. Boston City Hospital House Officers' Lecture. Undernutrition as a Clinical Problem. Dr. Garfield Duncan. New Cheever Amphitheater, Dowling Building, Boston City Hospital.

WEDNESDAY, MAY 11
*8:00–8:45 a.m. Case Presentations. Joslin Clinic. Joslin Auditorium, New England Deaconess Hospital.
*9:00–10:00 a.m. Surgical Grand Rounds. Massachusetts Memorial Hospitals.
10:30–11:15 a.m. Lecture on Diabetes for Doctors and Patients by a Member of the Joslin Clinic. Joslin Auditorium, New England Deaconess Hospital.
*11:00 a.m. Orthopedic Grand Rounds. Bigelow Amphitheater, White 3A, Massachusetts General Hospital.
*12:00 m.–1:00 p.m. Neurology, Neurosurgery and Psychiatry. Drs. Sullivan, B. Selverstone and Hope. Stearns Auditorium, New England Center Hospital.
*12:00 m.–1:00 p.m. Clinicopathological Conference. Jimmy Fund Building, Children's Medical Center, 35 Binney Street.
*12:00 m.–1:00 p.m. Roentgen Diagnostic Conference. 5th-floor teaching unit, Beth Israel Hospital.
*12:15 p.m. Vascular Rounds. Drs. Edward A. Edwards and Chilton Crane. D-Main, Peter Bent Brigham Hospital.
*12:30 p.m. Weekly Pathological Meeting. Joslin Auditorium, New England Deaconess Hospital.
12:30–1:30 p.m. Weekly Staff Review of Autopsies. Dr. William A. Meissner. Joslin Auditorium, New England Deaconess Hospital.
*1:00 p.m. Alcoholism Clinic. Peter Bent Brigham Hospital.
*1:00 p.m. Massachusetts General Hospital Hand Clinic. The Clinics of the Massachusetts General Hospital.
*2:00 p.m. Rectal Clinic. Peter Bent Brigham Hospital.
*2:00–3:00 p.m. Pediatric Conference for Practitioners. Jimmy Fund Building, Children's Medical Center, 35 Binney Street.
*4:00–5:30 p.m. Fracture Lecture. Boston City Hospital.
*4:00–6:00 p.m. Overholt Thoracic Conference. Joslin Auditorium, New England Deaconess Hospital.
*6:00 p.m. Alcoholism Clinic. Peter Bent Brigham Hospital.
*7:30–9:30 p.m. Lahey Clinic Lecture. Surgery of Tuberculosis. Dr. Norman Wilson. Joslin Auditorium, New England Deaconess Hospital.

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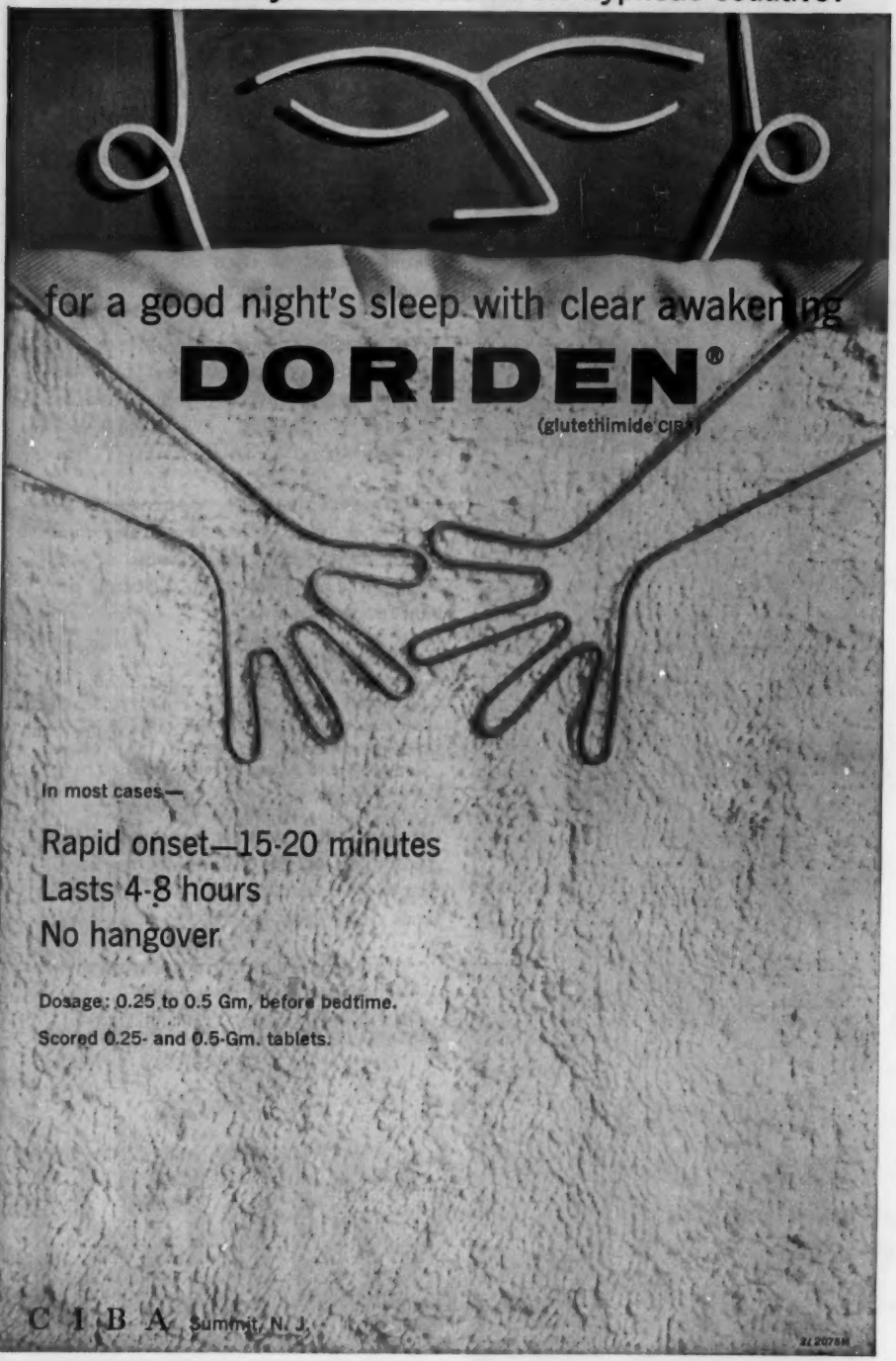

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